ESTIMATION OF SERUM CALCIUM LEVELS IN NEWLY DETECTED ESSENTIAL HYPERTENSIVE PATIENTS

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BONAFIDE CERTIFICATE

This is to certify that **" ESTIMATION OF SERUM CALCIUM LEVELS IN NEWLY DETECTED ESSENTIAL HYPERTENSIVE PATIENTS"** is a bonafide work done by **Dr. D. Raju,** post graduate student, Department of General Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in partial fulfillment of regulations of **The Tamilnadu Dr.M.G.R.Medical University** for the award of **M.D.Degree Branch I (General Medicine)** during the academic period from May 2007 to March 2010.

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CONTENTS

INTRODUCTION

Hypertension remains the leading cause of death worldwide and one of the world's great public health problems(WHO).[1] Affecting 1 billion people worldwide, systemic hypertension remains the most common, readily identifiable and reversible risk factor for myocardial infarction, stroke, heart failure, atrial fibrillation, aortic dissection and peripheral arterial disease. Because of escalating obesity and population aging in developed and developing countries, the global burden of hypertension is rising and projected to affect 1.5 billion persons, one third of the world's population, by the year 2025.[2]

Although our understanding of the pathophysiology of essential hypertension has increased, the etiology still remains hypothetical. Various studies have shown that essential hypertension is associated with disturbed calcium metabolism like increased cytosolic calcium and decreased serum calcium levels and also increased urinary excretion of calcium in patients with essential hypertension.[3-6]

In this study total serum calcium levels and corrected serum calcium levels of essential hypertension patients is compared and correlated with matched normotensive controls. Also the calcium levels are compared and correlated within the various subsets of hypertensive population viz.. age, sex, alcohol, smoking, lifestyle, BMI.

AIM OF THE STUDY

1. To compare the total and corrected serum calcium levels between patients with newly detected essential hypertension and normotensive controls.

2. To compare the total and corrected serum calcium levels of various subsets of patients with newly detected essential hypertension viz.. age, sex , smoking , alcohol, life style, BMI.

3. To determine the correlation of total and corrected serum calcium levels with systolic blood pressure in patients with essential hypertension.

4. To determine the correlation of total and corrected serum calcium levels with diastolic blood pressure in patients with essential hypertension.

REVIEW OF LITERATURE

BLOOD PRESSURE:

DEFINITION: [7,8]

Blood pressure is defined as the lateral pressure exerted by the column of blood against any unit area of the vessel wall. It is almost always measured in millimetres of mercury (mmHg).

CLASSIFICATION OF BLOOD PRESSURE: [9]

TABLE – 1

Pressure category	Systolic BP (mmHg)	Diastolic BP (mmHg)
Normal	< 120	< 80
Prehypertension	120 - 139	80 - 89
Hypertension		
Stage I	140-159	90 – 99
Stage II	>160	>100

GUIDELINES FOR MEASUREMENT OF BLOOD PRESSURE:[9,10]

PATIENT CONDITIONS:

I . Posture

1. Initially, particularly >65 years, with diabetes, or receiving antihypertensive therapy, check for postural changes by taking

readings after 5 min supine, then immediately and 2 min after standing.

2. For routine follow-up, the patient should sit quietly for 5 min with the arm bared and supported at the level of the heart and the back resting against a chair.

II . Circumstances

1. No caffeine or smoking within 30 min preceding the reading.
2. A quiet, warm setting.

III . Equipment

A . Cuff size

1. The bladder should encircle atleast 80% of the circumference and cover two-thirds of the length of the arm.
2. Various cuff sizes are available and one should choose appropriate cuff according to the individual and site of measurement. [11]

   Arm circumference 22 - 26 cm, 12 x 22 cm cuff (small adult)

   Arm circumference 27 - 34 cm, 16 x 30 cm cuff (adult)

   Arm circumference 35 - 44 cm, 16 x 36 cm cuff (large adult)

   Arm circumference 45 - 52 cm, 16 x 42 cm cuff (adult thigh)
3. A too small bladder may cause falsely high readings.

B . Manometer

1. Either a mercury, recently calibrated aneroid or validated electronic device.

C . Stethoscope

1. The bell of the stethoscope should be use
2. Avoid excess bell pressure.

IV . Infants

Use ultrasound (e.g., the Doppler method).

V .Technique

A . Number of readings

1. On each occasion, take atleast two readings, separated by as much time as is practical; if readings vary >5 mmHg, take additional readings until two are close.
2. For diagnosis, obtain three sets of readings at least 1 week apart.
3. Initially, take pressure in both arms; if the pressures differ, use the arm with the higher pressure.
4. If the arm pressure is elevated, take the pressure in one leg, particularly in patients <30 years old.

B . Performance

1. Inflate the bladder quickly to a pressure 20 mmHg above the systolic pressure, recognized by disappearance of radial pulse, to avoid an auscultatory gap.
2. Deflate the bladder 2 to 3 mmHg/second.
3. Record the Korotkoff phase I (appearance) and phase V (disappearance). Phase I corresponds to systolic pressure and phase V provides a better measure of diastolic blood pressure. Nevertheless, in those conditions where Korotkoff sounds remain audible despite complete deflation of the cuff (aortic regurgitation, arteriovenous fistula, pregnancy) phase IV (muffling) must be used for the diastolic measurement. [12]
4. If the Korotkoff sounds are weak, have the patient raise the arm and open and close the hand 5-10 times, then inflate the bladder quickly.

C **. Recordings**

Note the pressure, patient position, the arm, and cuff size (e.g., 140/90, seated, right arm, large adult cuff).

HYPERTENSION

DEFINITION:

Hypertension currently is defined as a usual BP of 140/90 mmHg or higher, BP levels for which the benefits of pharmacological treatment have been definitively established in randomized placebo-controlled trials.[2] Clinically, hypertension might be defined as that level of blood pressure at which the institution of therapy reduces blood pressure related morbidity and mortality. [13]

EPIDEMIOLOGY:

From an epidemiologic perspective, there is no obvious level of blood pressure that defines hypertension. In adults, there is a continuous, incremental risk of cardiovascular disease, stroke, and renal disease with increasing levels of both systolic and diastolic blood pressure.

The overall prevalence of hypertension varies from 6 – 32 % [14] thus affecting 1 billion population worldwide. In India the prevalence of hypertension is 59.9 and 69.9 per 1000 in males and females in urban population while it is 35.5 and 35.9 per 1000 in males and females in rural population respectively.[15]

Depending on etiology hypertension can be divided into two types.

1. Primary or essential hypertension.
2. Secondary hypertension.

PRIMARY (ESSENTIAL) HYPERTENSION:

DEFINITION :[16]

Primary hypertension, which accounts for 95 percent of all cases of hypertension, has been traditionally defined as high blood pressure for which an obvious secondary cause (e.g., renovascular disease, aldosteronism, pheochromocytoma, or gene mutations) cannot be determined.

Although primary hypertension is a heterogeneous disorder, some of the main causes of high blood pressure in primary hypertension are known. For example, overweight and obesity may account for as much as 65 to 75% of the risk for primary hypertension. Other factors, such as sedentary lifestyle, excess intake of alcohol or salt, and low potassium intake, are also known to increase blood pressure in many patients who are classified as having primary hypertension.

MECHANISMS OF PRIMARY(ESSENTIAL) HYPERTENSION :[2]

A multitude of neurohormonal , renal, and vascular mechanisms interact to varying degrees in contributing different hemodynamic forms of hypertension.

NEURAL MECHANISMS:

In young adults, primary hypertension consistently is associated with increased heart rate and cardiac output, plasma and urinary norepinephrine levels, regional norepinephrine spillover, peripheral postganglionic sympathetic nerve firing (by microelectrode recordings) and alpha-adrenergic receptor–mediated vasoconstrictor tone in the peripheral circulation.[17] Sympathetic overactivity has also been demonstrated in several other forms of established human hypertension like, hypertension associated with obesity, sleep apnoea, early type 2 diabetes mellitus and prediabetes, chronic kidney disease, heart failure and immunosuppressive therapy with calcineurin inhibitors such as cyclosporine. In these conditions, central sympathetic outflow can be driven by deactivation of inhibitory neural inputs (e.g., baroreceptors), activation of excitatory neural inputs (e.g., carotid body chemoreceptors, renal afferents) or by circulating angiotensin II, which activates pools of excitatory brain stem neurons that are devoid of a blood-brain barrier.

BARORECEPTORS:

In hypertension, the baroreceptors are reset to defend a higher level of BP. Baroreflex control of sinus node function is impaired even in mild hypertension but baroreflex control of systemic vascular resistance and BP is well preserved. Surgically-implanted carotid baroreceptor pacemakers

produce sustained BP reductions in dog models of hypertension[18] and parallel multicenter clinical trials are currently underway in patients with medically refractory hypertension.

Partial baroreceptor dysfunction is common in elderly hypertensives and typically presents with a triad of orthostatic hypotension, supine hypertension and symptomatic postprandial hypotension, the latter initiated by splanchnic pooling after carbohydrate-rich meals.[19]

RENAL MECHANISMS:

The kidney is the culprit and victim in hypertension, producing a vicious cycle of progressive renal dysfunction and hypertension. In many forms of experimental and human hypertension, the fundamental abnormality is an acquired or inherited defect in the kidney's ability to excrete the excessive sodium load imposed by a modern high salt diet. Renal sodium retention expands the plasma volume, increasing cardiac output and triggering autoregulatory responses that increase systemic vascular resistance. Salt retention also augments the smooth muscle contraction produced by all known endogenous vasoconstrictor substances.

Resetting of Pressure natriuresis:

In normotensive individuals, BP elevation invokes an immediate increase in renal sodium excretion to shrink plasma volume and return BP to

normal. In almost all forms of hypertension, the pressure-natriuresis curve is shifted to the right and, in salt-sensitive hypertension, the slope is reduced. Resetting of the pressure-natriuresis curve leads to nocturia, one of the most common and bothersome symptoms in patients with uncontrolled hypertension. Hypertensive individuals excrete the same amount of a given dietary sodium load as normotensive individuals, but at a higher BP, and require many more hours to excrete the sodium load and achieve sodium balance.

Low birth weight:

Low birth weight with reduced nephrogenesis increases the risk of developing adult salt-dependent hypertension. Adult hypertensives have fewer glomeruli per kidney but very few obsolescent glomeruli, suggesting that nephron dropout and decreased total filtration surface area is the cause and not the consequence of the hypertension .[20]

GENETIC CONTRIBUTIONS:

Animal and human studies have implicated an important genetic contribution to salt-sensitive hypertension. Rats with inbred defects in the kidney's ability to excrete sodium remain relatively normotensive on a sodium-restricted diet but become severely hypertensive when fed a high-sodium diet, a model of salt-sensitive hypertension that can be cured by

renal transplantation. A similar gene-environment interaction has been postulated to explain why persons of sub-Saharan African ancestry remain normotensive on a sodium-restricted diet in rural Africa but are predisposed to hypertension when exposed to a high-sodium western diet. However, sodium sensitivity of BP can be acquired without invoking any genetic contribution. With long-standing hypertension, BP becomes increasingly salt-sensitive as renal function declines in patients of all ethnic backgrounds.

VASCULAR MECHANISMS:

Alterations in the structure and function of small and large arteries play a pivotal role in the pathogenesis and progression of hypertension.

Endothelial dysfunction:

The endothelial lining of blood vessels is critical to vascular health and constitutes a major defence against hypertension. Dysfunctional endothelium is characterized by impaired release of endothelial-derived relaxing factors (e.g., NO, endothelium-derived hyperpolarizing factor) and enhanced release of endothelium-derived constricting, proinflammatory, prothrombotic and growth factors. The latter include endothelin, thromboxane and TGF-β .[21]

The endothelium of all blood vessels expresses the enzyme nitric oxide synthase (NOS), which can be activated by bradykinin or acetylcholine or by the cyclic laminar shear stress that accompanies hypertension, particularly with the widened pulse pressure in ISH. Once activated, NOS converts l-arginine to citrulline, an inert substance and nitric oxide (NO), a volatile gas that diffuses to the adjacent vascular smooth muscle and activates a series of G kinases that culminate in vasodilation .

In humans, endothelium-dependent vasodilation can be assessed by measuring increases in the large artery (forearm or coronary) diameter following intraarterial infusion of acetylcholine or release of ischemia (e.g., arrested forearm circulation) or a sudden elevation in BP (cold pressor test). Competitive inhibitors of NOS specifically block endothelium-dependent dilation but do not block the dilation of these arteries produced by exogenous nitrovasodilators (e.g., nitroglycerin, nitroprusside). These measurements most often are obtained with brachial artery ultrasound. More accurate measurements require invasive techniques.

C-reactive protein (CRP) is an easily measured serum biomarker for determining blood vessel inflammation and endothelial dysfunction.[22] Cross-sectional studies have shown strong correlations between elevated CRP and arterial stiffness and elevated pulse pressure. Longitudinal studies

have implicated elevated CRP levels as a risk factor for new onset of hypertension and accelerated progression of hypertensive target organ disease, possibly beyond that explained by BP elevation alone.

One of the principal mechanisms of endothelial cell dysfunction in hypertension is the production of superoxide anion and other reactive oxygen species that quench NO, thereby reducing its bioavailability. There are three main enzymatic sources of vascular superoxide:(1) NADPH oxidases, which are universally expressed in all vascular cell types and activated by circulating A II; (2) NOS, which produces superoxide only when an important cofactor (tetrahydrobiopterin) is deficient(NOS uncoupling); and (3) xanthine oxidase, which produces uric acid.[23]

Generation of reactive oxygen species by xanthine oxidase almost certainly accounts for the associations among elevated serum uric acid levels and hypertension .[24] However, uric acid itself is an antioxidant. Thus the xanthine oxidase inhibitor allopurinol does not consistently lower BP in patients with hypertension because the drug simultaneously reduces both these opposing effects on vascular function .[25]

Vascular remodelling:

Over time, endothelial cell dysfunction, neurohormonal activation and elevated BP cause remodelling of blood vessels, which further perpetuates the hypertension .[26] An increase in the medial thickness relative to lumen diameter (increased media-to-lumen ratio) is the hallmark of hypertensive remodelling in small and large arteries. Small artery remodelling is initiated by vasoconstriction, which normalizes wall stress and averts a trophic response. Normal smooth muscle cells rearrange themselves around a smaller lumen diameter; a process termed inward eutrophic remodelling. The media-to-lumen ratio increases but the medial cross-sectional area remains unchanged. By decreasing lumen diameter in the peripheral circulation, inward eutrophic remodelling increases systemic vascular resistance, the hemodynamic hallmark of diastolic hypertension.

In contrast, large artery remodelling is characterized by the expression of hypertrophic genes, triggering increases in medial thickness as well as the media-to-lumen ratio. Such hypertrophic remodelling involves not only an increase in the size of vascular smooth muscle cells but also an accumulation of extracellular matrix proteins such as collagen and fibronectin, because of activation of TGF- β. The resultant large artery stiffness is the hemodynamic hallmark of ISH.

Antihypertensive therapy may not provide optimal cardiovascular protection unless vascular remodelling is prevented or reversed by normalizing hemodynamic load, restoring normal endothelial cell function, and eliminating the underlying neurohormonal activation .[26]

HORMONAL MECHANISMS:

Renin-Angiotensin-Aldosterone System.

Activation of the renin-angiotensin-aldosterone system (RAAS) is one of the most important mechanisms contributing to endothelial cell dysfunction, vascular remodelling, and hypertension. Renin, a protease produced solely by the renal juxtaglomerular cells, cleaves angiotensinogen to Angiotensin I(A I), which is converted by angiotensin-converting enzyme to Angiotensin II(A II) . Chymase, a serine protease in the heart and systemic arteries, provides an alternative pathway for conversion of Angiotensin I to Angiotensin II. The interaction of A II with G protein–coupled AT_1 receptors activates numerous cellular processes that contribute to hypertension and accelerate hypertensive end-organ damage. These include vasoconstriction, generation of reactive oxygen species, vascular inflammation, vascular and cardiac remodelling, and production of aldosterone, the principal mineralocorticoid. There is increasing evidence that aldosterone, A II, and even renin and prorenin activate multiple signaling pathways that can damage vascular health and cause hypertension.

Aldosterone and ENaC Regulation:

RAAS activation is a major homeostatic mechanism to counter hypovolemic hypotension (as with hemorrhage or salt and water deprivation). Interaction of aldosterone with cytosolic mineralocorticoid receptors in the renal collecting duct cells recruits sodium channels from the cytosol to the surface of the renal epithelium. The epithelial sodium channels (ENaCs) so recruited increase sodium reabsorption, thereby reexpanding plasma volume. Conversely, modern high-salt diets should engender continual feedback inhibition of the RAAS. Suppression of serum aldosterone should trigger sequestration of ENaCs by endocytosis and increased renal sodium excretion, thereby shrinking plasma volume to protect against salt-sensitive hypertension.

Thus, in the setting of high dietary sodium and elevated BP, the RAAS should be completely suppressed and any degree of RAAS activity is inappropriate. In normotensive individuals, the risk of developing hypertension increases with increasing levels of serum aldosterone that are well within the normal range .[27] In African Caribbean hypertensives, serum aldosterone levels are higher than in white hypertensives despite lower plasma renin levels [28], implicating abnormal aldosterone production by renin-independent mechanisms, a forme fruste of primary aldosteronism. By stimulating mineralocorticoid receptors in the heart and kidney, circulating

aldosterone may contribute to the development of cardiac and renal fibrosis in hypertension .[29] By stimulating mineralocorticoid receptors in the brain stem, aldosterone also may contribute to sympathetic overactivity.

Receptor mediated actions of angiotensin II:

Two main types of angiotensin receptors are known. AT1 receptors are widely expressed in the vasculature, kidney, adrenals, heart, liver and brain. AT 1 receptor activation explains most of the hypertensive actions of A II. Furthermore, constitutes a major therapeutic target for interrupting every step in cardiovascular disease progression, from vascular remodelling and formation of atherosclerotic plaque to stroke, myocardial infarction (MI) and death. In contrast, AT_2 receptors are widely distributed in the fetus but in adults, are found only in the adrenal medulla, uterus, ovary, vascular endothelium and distinct brain regions. In rodents, AT_2 receptor activation opposes some of the deleterious effects of AT_1 receptors by promoting endothelium-dependent vasodilation by bradykinin and nitric oxide pathways. However, recent animal studies have suggested that AT_2 receptors can be profibrotic but their role in human hypertension remains speculative

Receptor mediated actions of renin and prorenin:

In the traditional RAAS, prorenin has been considered to be the inactive precursor of renin, which functions solely to generate A I by

enzymatic cleavage of angiotensinogen. These concepts are rapidly evolving as newer studies implicate prorenin and renin as direct cardiac and renal toxins. Prorenin is inactive because a 43–amino acid hinge is closed and prevents it from binding to angiotensinogen. In the kidneys, inactive prorenin is converted to active renin when this inhibitory hinge region is enzymatically cleaved. When circulating prorenin binds to a newly discovered (pro)renin receptor in the heart and kidneys, the hinge is opened (but not cleaved), and this nonenzymatic process fully activates prorenin .[30,31] As a result, TGF- β production is accelerated, leading to collagen deposition and fibrosis. This receptor-mediated process is completely independent of A II generation and therefore unaffected by angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs). Although ACEIs and ARBs are excellent antihypertensives , they trigger large reactive increases in prorenin and renin production that may counter some of the cardiovascular protection afforded by reduced AT_1 receptor activation.

OTHER MECHANISMS:

Heredity:

Genetic factors have long been assumed to be important in the genesis of hypertension. This view is supported by animal studies as well as population studies. One approach has been to assess the correlation of blood

pressure within families (familial aggregation) .[32] From these studies, the minimum size of the genetic factor can be expressed by a correlation coefficient of approximately 0.2. However, the variation in the size of the genetic factor in different studies reemphasizes the likely heterogeneous nature of the essential hypertensive population. Most studies support the concept that the inheritance is probably multifactorial. Now monogenic defects have been reported which have one of their consequences, an increased arterial pressure[33] e.g. Liddle's syndrome. Suseptibility genes have now been reported [34] e.g. Angiotensinogen gene and α – adducing gene.

Environment :[32]

A number of environmental factors have been implicated in development of hypertension, including salt intake, obesity, stress, alcoholism, family size and crowding. These factors all have been assumed to be important in the rise in blood pressure with age in more affluent societies, in contrast to the decline in blood pressure with age in more primitive cultures.

Insulin resistance:

Insulin resistance and/or hyperinsulinemia have been suggested as being responsible for the increased arterial pressure in some patients with hypertension. Insulin resistance is common in patients with type 2 diabetes

mellitus or obesity.[35] A diminished activity of calmodulin-stimulated Ca^{2+}-ATPase despite increased levels of insulin, a known activator of this pump, further suggesting the presence of insulin resistance in normotensive offsprings of essential hypertensive individuals. Since Ca^{2+}-ATPase is an extrusion pump, a drop in its activity may lead to an increase in intracellular calcium accumulation and thus contribute to the development of hypertension.[36] Also hyperinsulinemia causes renal sodium retention and increases sympathetic activity. It also causes smooth muscle hypertrophy due to mitogenic action of insulin.

OVERVIEW OF PATHOGENESIS :[10]

Hundreds of papers have been published describing the work of thousands of investigators who have spent many millions, perhaps billions, of dollars, yet the pathogenesis of primary hypertension is as elusive and enigmatic as ever. The pressure required to move blood through the circulatory bed is provided by the pumping action of the heart [cardiac output (CO)] and the tone of the arteries [peripheral resistance (PR)]. Each of these primary determinants of BP is, in turn, determined by the interaction of the exceedingly complex series of factors displayed in part in figure 1.

Hypertension has been attributed to abnormalities in virtually every one of these factors. Each will be examined and attempts will be made along

the way to integrate them into logical hypotheses. It is unlikely that all these factors are operative in any given patient; but multiple hypotheses may prove to be correct, because the hemodynamic hallmark of primary hypertension - a persistently elevated vascular resistance, may be reached through a number of different paths. Before the final destination, these may converge into either structural thickening of the vessel walls or functional vasoconstriction. Moreover, individual factors often interact and the interactions are proving to be increasingly complex.

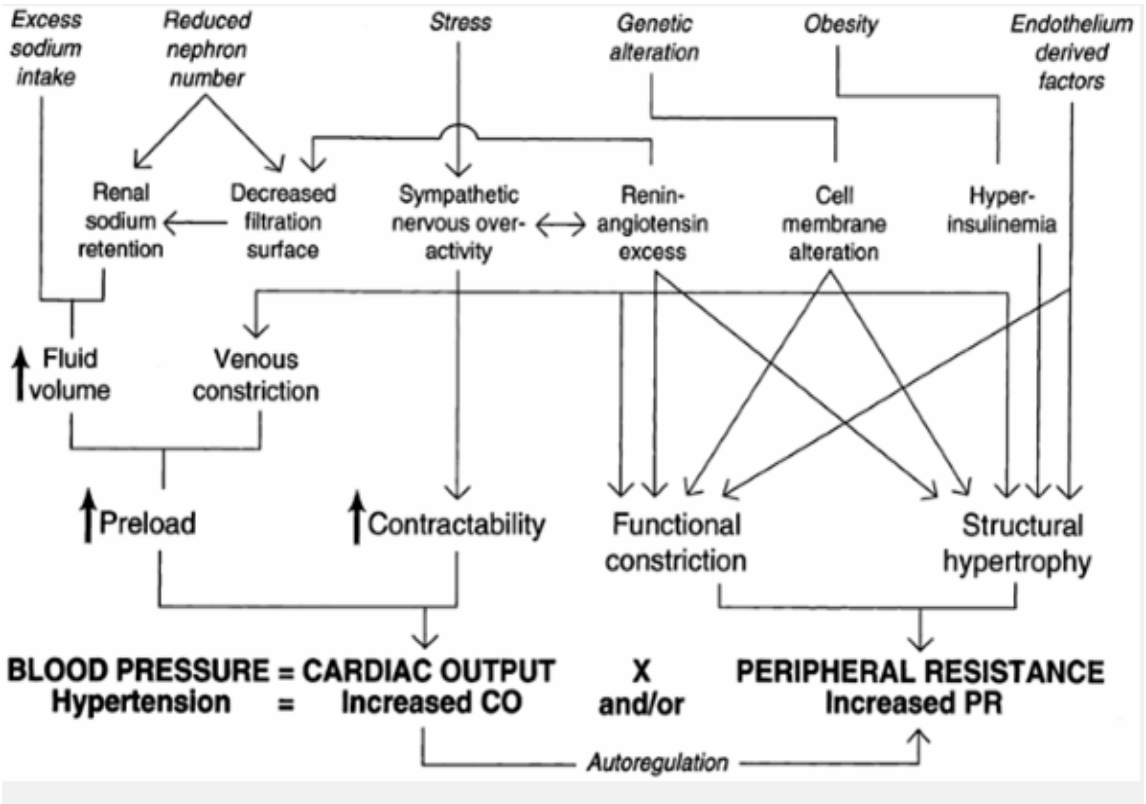


Figure 1. Pathogenesis of Primary Hypertension .

We will follow the outline shown in Figure 1, recognizing that the position of each factor in the outline is not necessarily in the order that the

hemodynamic cascade follows in the pathogenesis of hypertension. Without knowing what starts the process, we can lay out only a preliminary blueprint that can be used by beginning at multiple sites.

Risks Influencing Prognosis in Patients with Hypertension :[37]

Risk Factors for Cardiovascular Disease:

Levels of systolic and diastolic blood pressure

Age (yr) — men > 55; women > 65

Smoking

Dyslipidemia

Family history of premature cardiovascular disease

Abdominal obesity

Diabetes mellitus

CRP ≥1 mg/dl

Subclinical Target Organ Damage:

Left ventricular hypertrophy

Ultrasound evidence of arterial wall thickening or atherosclerotic plaque

Estimated glomerular filtration rate ≤60 ml/min/1.73 m^2

Microalbuminuria

Clinical Target Organ Damage:

Cerebrovascular disease

Ischemic stroke

Cerebral hemorrhage

Transient ischemic attack

Heart disease

Myocardial infarction or acute coronary syndrome

Angina

Coronary revascularization

Congestive heart failure

Renal disease

Diabetic nephropathy

Chronic kidney disease

Proteinuria (>300 mg/24 hr)

Peripheral arterial disease

Advanced retinopathy

Hemorrhages or exudates

Papilledema

SECONDARY HYPERTENSION :[38]

In 5 – 10% of patients the causes of hypertension can be identified, and some are curable. By definition, these are designated as secondary hypertension.

CAUSES OF SECONDARY HYPERTENSION:[38 , 39]

Renal disorders:

Renal parenchymal

- ❖ Acute and chronic glomerulonephritis; pyelonephritis; nephrocalcinosis; neoplasms; glomerulosclerosis; interstitial, hereditary or radiation nephritis.
- ❖ Obstructive uropathies and hydronephrosis
- ❖ Renin-secreting renal tumors (hemangiopericytoma, Wilms or renal cell, pancreatic, ovarian tumors)
- ❖ Congenital defect in renal Na transport (Liddle's syndrome)
- ❖ Renal trauma

Renovascular

- Renal arterial lesions, occlusions, stenoses, aneurysms, thromboses
- Renal vasculitis or glomerulitis
- Coarctation of the aorta with renal ischemia
- Aortitis with renal ischemia

Adrenocortical disorders:

- ❖ Cushing syndrome (cortisol excess)
- ❖ Primary aldosteronism due to adenoma (Conn syndrome)

- ❖ Pseudoprimary aldosteronism (bilateral adrenocortical hyperplasia)
- ❖ Congenital or acquired enzymatic defects with excess Na^+-retaining steroids (11β-hydroxylase deficiency; 11β-hydroxysteroid dehydrogenase deficiency, 17α-hydroxylase deficiency)
- ❖ Adrenal carcinoma
- ❖ Ectopic corticotropin-secreting tumor

Pheochromocytoma (adrenal medullary or extraadrenal chromaffin tumors)

Other endocrine causes:

- ❖ Hypothyroidism (diastolic hypertension)
- ❖ Hyperthyroidism (systolic hypertension)
- ❖ Hypercalcemic states, hyperparathyroidism
- ❖ Acromegaly

Toxemias of pregnancy

Neurogenic factors:

- ❖ Increased intracranial pressure
- ❖ Familial dysautonomia
- ❖ Acute porphyria, buffer denervation, poliomyelitis, spinal cord injuries
- ❖ Psychogenic

Iatrogenic and other causes:

- Oral contraceptive or estrogen therapy
- Mineralocorticoid or glucocorticoid therapies, licorice ingestion (i.e., acquired 11β-hydroxysteroid dehydrogenase deficiency)
- Sympathomimetic drugs (decongestant)
- Antidepressants
- Alcohol abuse
- Lead toxicity
- Monoamine oxidase inhibitors (interactions with other agents)
- Excessive salt appetite

ROLE OF CALCIUM IN ESSENTIAL HYPERTENSION:

There are several hypothesis that abnormalities of calcium homeostasis at both an organ and cellular level as a primary factor in the pathogenesis of human and experimental hypertension .Also a low calcium intake has been associated with increase in blood pressure in several epidemiological studies.

An increase in leucocyte cytosolic calcium levels has been reported in some hypertensives, similarly an increase in RBC and platelet cytosolic calcium levels has also been reported in some hypertensives.[40] Several

studies have reported a potential link between the salt sensitive forms of hypertension and calcium.

It has been postulated that with salt loading and defect in the kidney's ability to excrete , a secondary increase in circulating natriuretic factors may occur .One of these, the digitalis like natriuretic factor inhibits ouabain sensitive Na^+-K^+ATPase causing intracellular calcium accumulation and hyperreactive vascular smooth muscle.[41,42]

In one third of patients with hyperparathyroidism, hypertension is noted and can be attributed to renal parenchymal demage due to nephrolithiasis and nephrocalcinosis. However increased calcium levels also have a direct vasoconstrictive effect. In some cases hypertension can be treated by correcting hypercalcemia.[43] Additional studies are needed to resolve these seemingly conflicting observation.

CALCIUM IN PATHOGENESIS OF PRIMARY HYPERTENSION:

The calcium status of humans with essential hypertension and genetic animal models of hypertension is characterized by low serum ionized calcium concentration, increased urinary calcium excretion, and increased parathyroid hormone (PTH) concentration. Calcitriol metabolism and bone mineralization are also altered in hypertension. These alterations in systemic calcium metabolism may be linked to factors responsible for the elevated

blood pressure. Cytosolic free calcium tends to be increased in most cells that have been studied from hypertensive humans and animals. Changes in cellular calcium metabolism may be partly mediated by calcium-regulating hormones that tend to be elevated in essential hypertension such as PTH and calcitriol.[44] This raised levels may be due to an intrinsic defect in renal calcium handling.[45] Administration of supplemental dietary calcium tends to suppress PTH, calcitriol, and intracellular free calcium.

In hypertensive patients there is a defect in excreting the digitalis like natriuretic factor which inhibits ouabain sensitive Na^+-K^+ATPase causing intracellular sodium accumulation. This increased intracellular sodium causes intracellular calcium accumulation in the vascular smooth muscle cells leading to an increase in contractility and vascular tone, resulting in increased peripheral resistence and blood pressure.[42]

There exists numerous relationship between calcium and sodium as stated by Blaustein.[46]

a. Inhibition of N^+-K^+ Exchange pumps would depolarise muscle fibres and thereby increase calcium entry through voltage sensitive calcium channels.

b. An increase in intracellular sodium will result in a sodium electrochemical gradient between sarcoplasm and external medium causing reduced extrusion of calcium from the cell.
c. An increase in intracellular sodium in the presynaptic terminal of sympathetic neurons promoting calcium dependant noradrenaline release causing release of calcium from cellular stores.
d. A very small rise in intracellular sodium, theoretically is adequate to cause enough rise in intracellular calcium, to increase the resting vascular smooth muscle tone by 50%.

Defects of Calcium at Cellular level in Humans:

Several defects in cellular calcium concentration, membrane binding, and transport kinetics have been identified in red blood cells (RBCs), platelets, and adipocytes of persons with essential hypertension, including reduced calcium buffering and sodium-calcium exchange. In RBCs of hypertensive persons, the amount of calcium bound to the inside surface is reduced by 25 to 30%.[47,48] This decrease in membranebound calcium has been linked to increased permeability of the cell to sodium and partial inhibition of Na^+-K^+ ATPase activity.

While calmodulin content and distribution in RBCs from hypertensive subjects have been found to be normal, the ability of calmodulin to activiate Ca^{2+}-ATPase is impaired, since the affinity of the calcium pump for calcium is reduced and the maximal activity of the pump is lower.[49] It was also demonstrated that basal (unstimulated) Ca^{2+}-ATPase activity is decreased in RBC membranes of hypertensive subjects compared with that in matched control subjects.[50] A diminished Ca^{2+}-ATPase or calmodulin stimulated Ca^{2+}-ATPase has been demonstrated in platelets and erythrocytes obtained from primary hypertensive subjects.[51] The collective observations suggest a fundamental alteration in the hypertensive subject's inner membrane calcium-calmodulin binding kinetics and subsequent activation of energy dependent cation pumps such as Ca^{2+}-ATPase.[52]

Intracellular free calcium concentrations are increased in platelets of subjects with high blood pressure. Those with higher intracellular free calcium concentrations were associated with lower extracellular calcium levels, which demonstrates the dichotomy that can exist between these calcium pools. Calcium homeostasis of platelets is dependent on limiting membrane fluxes, stimulating efflux and promoting sarcoplasmic reticulum sequestration,[53] all being calcium- calmodulin- dependent processes.

Since Ca^{2+}-ATPase is of fundamental importance in the final regulation of normal cytosolic calcium content and if the RBC defects in

calcium binding and Ca^{2+}-ATPase activation are reflective of a more generalized membrane-associated defect, then it follows that intracellular free calcium would be modestly elevated in platelets. The possibility that a generalized defect does exist is supported by two additional observations: the intracellular calcium concentrations are elevated in RBCs of hypertensive persons [54] and adipocytes obtained from hypertensive subjects exhibit similar alterations of intracellular calcium.[55]

Effects of reduced calcium intake:

Various studies showed an association between dietary calcium intake and blood pressure however, the potential benefit of treating hypertension with increased dietary intake of calcium remain controversial. Previous analyses of the NHANES I and II data have yielded conflicting findings regarding the influence of various dietary variables on blood pressure, particularly calcium.[56] Many cross-sectional studies have shown relations between dietary calcium intake and blood pressure.[57,58] Data from the Western Electric Heart Study [59] showed that calcium intake was inversely related to the incidence of elevated DBP (95 mmHg or greater) but not of elevated SBP (160 mmHg or greater). A recent report of a large cohort study of women showed that dietary calcium intake was inversely related to hypertension among women.[60]

Increased urinary calcium excretion:

The total and fractional urinary calcium excretion is elevated in subjects with essential hypertension.[6] Consistent with these reports is a population survey that demonstrated a positive correlation between urinary calcium excretion and blood pressure among 9321 men.[61] Although dietary calcium was not measured in the survey, the greater urinary calcium excretion was thought to be reflective of a greater dietary intake of calcium. The likelihood of this cause-and effect relationship's holding is inconsistent with the epidemiological data summarized above, which report decreased intake of calcium by hypertensive persons.

In addition, in an intervention trial, which assessed urinary calcium and dietary calcium intake in hypertensive subjects had observed that lower intakes were associated with higher excretion rates.[62] Whether the excessive urinary calcium excretion reflects an enhanced intestinal absorption of a decreased oral intake or a decreased ability of the kidney to reabsorb the cation is controversial. However the pattern of increased PTH levels and urinary cyclic AMP, lower serum ionized calcium values and reduced serum phosphorus levels appears to argue strongly against the former and in favour of the latter possibility.

Calcium supplements:

In 42 mostly short-term studies of either calcium supplements (33) or dietary intervention (9) in 4560 nonpregnant adults, the blood pressure reduced by 1.44/0.84 mmHg.[63] Because calcium supplements sometimes raise blood pressure and increase the risk of kidney stones, the best course is to ensure that calcium intake is not inadvertently reduced by reduction of milk and cheese consumption in an attempt to reduce saturated fat and sodium intake.

Supporting literatures :

Some epidemiological studies are discussed here to enlighten the present study.

K. Sudhakar et al.,[5] in their study enrolled one hundred and seventeen confirmed untreated essential hypertensive patients referred from cardiology unit of Osmania University General Hospital and Osmania University Health Centre, Hyderabad and their 77 first degree relatives (33 siblings and 44 offsprings). One hundred and sixty persons with normal BP and without family history of hypertension were taken as normotensive age and sex matched controls.The mean total serum calcium levels in males and females were significantly decreased in hypertensive group when compared with normotensive controls. In the first-degree relatives also the total serum

calcium levels were significantly decreased when compared with the controls.

AR Folsom et al.,[64] studied the serum calcium fractions in essential hypertensive and matched normotensive subjects. In the study, the hypertensive group comprised 28 subjects whose diastolic blood pressure on both occasions was greater than 90 mm Hg and who were not taking antihypertensive medication. One normotensive control was matched to each hypertensive subject. Controls were required to be of the same race, age and sex as the matched hypertensive subject. Hypertensive subjects had lower mean serum levels of ultrafilterable calcium, and ionized calcium.Calculated serum concentrations of complexed calcium were significantly lower in hypertensive subjects, while proteinbound calcium concentrations were higher. Serum phosphorus,serum albumin and dietary calcium intake, were not different between the two groups.

Fu, Y., Wang, S.,et al[65] assessed the relationships between plasma and intracellular Ca^{2+} ,Mg^{2+} and blood cell membrane ATPase activity in normotensive and hypertensive subjects in their study, which suggested that the hypertensive group consistently demonstrated a significant decreased activity of ATPase along with significantly lower plasma calcium and higher cytosolic calcium levels when compared with those in

normotensive group. No significant differences were found in either plasma magnesium or intracellular magnesium level between the two groups. This study conducted in China included 55 patients with essential hypertension and 32 normotensive controls.

Strazzullo P et al.,[66] Erne P., Bolli P., et al.,[67] Touyz, R.M.,et al.,[4] also reported a decrease in the total serum calcium concentration in essential hypertensive patients in their corresponding studies.While McCarron DA.,[68] and Resnick LM, Laragh JH.,et al[69] noted that essential hypertensive subjects had a lower serum ionized calcium concentrations , compared with normotensive subjects, even when total calcium levels were similar.

Grobbee DE et al.,[70] in his research article "Calcium metabolism and familial risk of hypertension" stated that there is circumstantial evidence that disturbances of calcium metabolism are implicated in primary hypertension. From a large number of epidemiological studies, data have shown that a low dietary calcium intake increases the risk for high BP. There is no general sensitivity for the effects of inadequate calcium intake,but subgroups of hypertensive patients have been described, characterized by reduced serum ionized calcium levels, increased urinary excretion of calcium, raised intracellular calcium levels, reduced cellular membrane calcium binding, and other indicators of a relative calcium need. Some of these changes, however, may be secondary to blood pressure elevation.The family history

approach enables to study the pathophysiology of early primary hypertension, at a stage at which BP differences between future hypertensive subjects and normotensive subjects are still limited.

He also stated that in the "Dutch Hypertension and Offspring Study", young normotensive subjects were selected on the basis of presence or absence of familial predisposition for hypertension. The findings showed that disturbances in calcium metabolism were present in the early phase of primary hypertension and may precede the development of high BP. Moreover, they suggested that changes in calcium metabolism may be a characteristic of familial hypertension and could reflect a genetic basis for calcium sensitive hypertension. The presence of a relatively reduced serum calcium and increased plasma PTH level in the offspring of hypertensive parents indicates that calcium balance in prehypertensive subjects is maintained at a higher level of circulating PTH.

Oshima T et al.,[71] in his research article "Systemic and cellular calcium metabolism and hypertension" which he submitted to 'The Seminars in Nephrology 1995' mentioned that the calcium status of humans with essential hypertension and genetic animal models of hypertension is characterized by low serum ionized calcium concentration, increased urinary calcium excretion, and increased PTH concentration. These alterations in

systemic calcium metabolism may be linked to factors responsible for the elevated blood pressure. Cytosolic free calcium tends to be increased in most cells that have been studied from hypertensive humans and animals. Changes in cellular calcium metabolism may be partly mediated by calcium-regulating hormones that tend to be elevated in essential hypertension such as PTH and calcitriol. Administration of supplemental dietary calcium tends to suppress PTH, calcitriol, and intracellular free calcium. Further research is need concerning the observed association among systemic markers of calcium metabolism, cellular calcium metabolism, and arterial blood pressure regulation.

Various studies had showed an association between dietary calcium intake and blood pressure but the potential benefit of treating hypertension with increased dietary intake of calcium remain controversial. However there are certain populations which can be benefited from calcium supplementation.

Hojo, M.et al.,[72] in his study mentioned approximately 10% of pregnancies are accompanied by HT, pre-eclampsia accounting for 50% cases . Women at risk of developing PIH are typically responsive to increased calcium intake, with the incidence of hypertension being reduced

upto 40-50% in those with a 1500-2000 mg/day intake (there is an increased daily calcium requirement due to fetal processes).

Patients with salt-sensitive hypertension represent another group for whom increased calcium intake appears to be highly beneficial. Responses in blood pressure to salt ingestion have been shown to additionally depend on the adequacy of dietary mineral intake (calcium, magnesium, and potassium). In fact, in the NHANES I database, several individuals with high sodium intake were observed to have lower blood pressures than would be expected; upon further analysis it was found that the calcium and potassium intake of these individuals was high, revealed in a study by McCarron, D.A.et al.,[73]

MATERIALS AND METHODS

This study was carried out in Department of General Medicine at Government Kilpauk Medical College & Hospital,Chennai – 10 during the period between January 2009 and October 2009.This study was ethically approved by the Ethical committee of Government Kilpauk Medical College and Hospital, Kilpauk, Chennai – 10.

This study is a cross sectional study, with cases and controls enrolling 100 newly detected essential hypertension patients as cases and 50 suitable healthy individuals with normal blood pressure as controls, as per JNC-7 guidelines. Cases were selected from those who visited hypertension clinic and those who were admitted in wards during the study period. Controls were selected from those who attended medical outpatient department for minor ailments and healthy volunteers.

INCLUSION CRITERIA:

1. Newly detected essential hypertension patients.

EXCLUSION CRITERIA:

1. Essential hypertension.
    a. Known cases of hypertension.
    b. Patients on antihypertensive agents.

c. Patients with malignant hypertension.

2. Chronic renal failure.
3. Diabetes mellitus.
4. Ischemic heart disease.
5. Patients on hypertension inducing drugs.
6. Other causes of secondary hypertension.
7. Congestive cardiac failure.
8. Cerebrovascular accident.
9. Peripheral vascular disease.
10. Patients with acute illness.
11. Patients with any medical or surgical complications.
12. Adolescents and young adults < 30 yrs.
13. BMI < 18.5Kg/m^2.

Applying these criteria,100 essential hypertensive patients were selected and included in the study after informed consent (Group A). Similarly 50 normotensive controls were selected and involved in the study after informed consent(Group B).

Secondary hypertension patients were excluded from the study by detailed history and clinical examination and also with relevant haematological, urinary and radiological investigations.

METHODOLOGY

A detailed history taking and clinical examination was done in patients with particular reference to hypertension. Relevant urine and blood investigations were done. Fundus examination for hypertensive retinopathy and ultrasonogram were done in all patients.

1. Socio-demographic data:

Age Sex Occupation/ Income

Family H/o hypertension (Father/Mother/Siblings)

Life style(Sedentary if physical activity < 3 METS)

Alcohol(Alcoholic defined > 2 drinks / day)

Smoking (Smoker defined > 5 cigarettes or beedis / day)

2. Clinical data:

BMI:[77]

Body Mass Index(BMI) = Weight (kg) / Height (m) ^{2}.

Values 18.5 - 22.9 kg / m^2 was taken as normal weight.

23- 24.9 kg / m^2 was taken as overweight.

$\geq$ 25 kg / m^2 was taken as obesity.

Measurement of BP:

BP was measured as per JNC – 7 guidelines. Subjects were instructed not to take caffeine or smoking within 30 min preceding the

reading and were seated quietly for 5 min in a quiet room after emptying the bladder, with the arm bared and supported at the level of the heart and the back resting against a chair. A mercury manometer with appropriate cuffsize was used to measure the blood pressure. Korotkoff sounds phase I (appearance) was taken as systolic BP while phase V (disappearance) was taken as a measure of diastolic blood pressure. Two sets of BP readings were taken 30 min apart in both arms in sitting posture; if the pressures differ the arm with the higher pressure was taken. Lower limb BP was also taken in patients less than 40 years of age while BP recordings for postural hypotension were measured for those who aged more than 60 years.

Other vital signs

General & Systemic examination

3. Laboratory data:

Urinalysis:

Urine sample was collected for urine routine analysis which included sugar, protein, cytology and urinary sediments

Urine spot PCR:

Urine sample was collected to estimate protein creatinine ratio. Sulfo salicylic precipitation method was used for protein estimation.

Blood sugar:

Blood sugar was estimated by Trinder's (Glucose oxidase) method and read at 505/670 nm.

Renal function test:

The blood urea in this study was estimated using DAM method (Diacetyl Monoxime). Serum creatinine was estimated using Modified Jaffe's method. Electrolytes were measured by absorption spectrophotometry.

Chest X – ray :

ECG:

USG Abdomen:

ESTIMATION OF SERUM CALCIUM :[74]

Arsenazo III Method:

The principle of this method is that calcium bind specifically with arsenazo III at an acidic pH to form a blue – purple coloured compound. The intensity of the colour of the compound is directly proportional to the amount of calcium present in the sample. In the calcium estimation kit, there will be a standard sample with measured calcium of 10 mg/dl with which a standard stock solution(S) has to be prepared. The serum of patient

is noted as test sample (T). A solution with distilled water is also prepared and branded as blank (B). Now the absorbance (Abs) of standard(S) and test(T) are measured against the blank(B) by absorption spectrophotometry at 650 nm(Red) in room temperature within one hour and the readings to be noted. This method has a linearity upto 15 mg/dl.

Serum Calcium (mg/dl) = [Abs of (T) / Abs of (S)] x10.

ESTIMATION OF SERUM PHOSPHORUS:

Unreduced phosphomolybdate Method:[74]

The principle of this method is that phosphate ions in acidic medium react with ammonium molybdate to form a phosphomolybdate complex.This complex has an absorbance in the ultraviolet range (340nm).The intensity of the colour of the complex formed is directly proportional to the amount of inorganic phosphorus present in the sample. In the phosphorus estimation kit, there will be a standard sample with measured phosphorus of 5 mg/dl with which a standard stock solution(S) has to be prepared.The serum of patient is noted as test sample (T). A solution with distilled water is also prepared and branded as blank (B). Now the absorbance (Abs) of standard(S) and test (T) are measured against the blank (B) by absorption spectrophotometry at 340nm in room temperature within one hour and the readings to be noted. This method has a linearity upto 20 mg/dl.

Serum Phosphorus (mg/dl) = [Abs of (T) / Abs of (S)] x 5.

ESTIMATION OF SERUM ALBUMIN:[74]

Albumin BCG Method:

The principle of this method is that albumin bind with the dye bromocresol green (BCG) in a buffered medium to form a green coloured complex. The intensity of the colour of the complex formed is directly proportional to the amount of albumin present in the sample. In the albumin estimation kit, there will be a standard sample with measured albumin of 4g/dl with which a standard stock solution(S) has to be prepared. The serum of patient is noted as test sample (T). A solution with distilled water is also prepared and branded as blank (B). Now the absorbance (Abs) of standard(S) and test (T) are measured against the blank(B) by absorption spectrophotometry at 628 nm(Red) and the readings to be noted. This method has a linearity upto 7 g/dl.

Serum Albumin (g/dl) = [Abs of (T) / Abs of (S)] x 4.

CORRECTED SERUM CALCIUM LEVEL :[74,75,76]

= Serum Calcium(mg/dl) + { 0.8 [4.0 – Serum albumin (g/dl)] }

In this study the correction was done for albumin using the above formula and corrected serum calcium levels were obtained.

4. Statistical analysis:

Data was entered in Microsoft excel spread sheet and analysed statistically using SPSS software version 11.5.

Results were considered significant if the ‘p’ value was below 0.05.

The following tests were used for statistical analysis:

1. Chi – square test.
2. Student – t test.
3. Analysis of variance(ANOVA).
4. Pearson’s correlation.

RESULTS:

- A total of 150 individuals were selected . They were divided into two groups, group A and group B.
- Group A was the study group which included 100 newly detected essential hypertensive patients . Group B was the control group which included 50 normotensive individuals.
- In Group A, 52 (52%) were males and 48(48%) were females and in Group B, 26(52%) were males and 24(48%) were females.
- The mean age in group A was 55.52± 10.782 years and the mean age in group B was 52.06 ± 10.88 years.
- Majority of hypertensives in the study group were asymptomatic (46%), and among the symptomatic individuals the commonest symptom was giddiness (24%) followed by headache (10%) .
- In Group A, 70 (70%) were nonsmokers and 30 (30%) were smokers and in Group B,33(66%) were nonsmokers and 17 (34%) were smokers. In Group A, 69 (69%) were nonalcoholics and 31 (31%) were alcoholics and in Group B, 32(64%) were nonalcoholics and 18 (36%) were alcoholics.
- 52% (52) patients in group A had a sedentary lifestyle.
- The prevalence of family history of hypertension among hypertensive individuals was 37% (37).

- In group A, the prevalence of obesity as per India reworks obesity guidelines was 51% (51).
- The mean systolic blood pressure of group A and group B were 169.2800 ± 15.81847 mmHg and 111.7200 ± 6.88103mmHg while the mean diastolic blood pressure were 101.4000± 8.61054 mmHg and 71.1600 ±6.31280 mmHg respectively.
- The mean total serum calcium and corrected serum calcium levels in group A were 8.9160 ± 0.62529 mg/dl and 8.8263 ± 0.6525 mg/dl while the mean total serum calcium and corrected serum calcium levels in group B were 9.7042± 0.79350mg/dl and 9.6550 ± 0.80053 mg/dl respectively. The calcium levels were significantly lowered in group A when compared with group B.
- There was a negative correlation noted between the total and corrected serum calcium levels as against the systolic blood pressure.
- There was no correlation noted between the total and corrected serum calcium levels as against the diastolic blood pressure.
- There was no significant difference in the total and corrected serum calcium levels with age , sex , BMI , life style , smoking , alcohol , family history of hypertension in newly detected essential hypertensive patients.

AGE DISTRIBUTION BETWEEN GROUP A & GROUP B
TABLE – 2:

AGE GROUP(years))	GROUPS		Total
	A	B	
< 40	3 (3.0%)	2 (4.0%)	5 (3.3%)
41 – 50	33 (33.0%)	17 (34.0%)	50 (33.3%)
51 – 60	32 (32.0%)	17 (34.0%)	49 (32.7%)
61 – 70	21 (21.0%)	9 (18.0%)	30 (20.0%)
>70	11 (11.0%)	5 (10.0%)	16 (10.7%)
Total	100 (100.0%)	50 (100.0%)	150 (100.0%)

TABLE – 3:

GROUP	N	Mean Age	Std.	Std. Error
A	100	55.5200	10.78203	1.07820
B	50	55.0600	10.85791	1.53554

P value betweeen groups was 0.988. statistically not significant.

There was no statistically significant difference noted in age distribution between the groups A & B. (P > 0.05)

SEX DISTRIBUTION BETWEEN GROUP A & GROUP B :

TABLE – 4:

SEX	GROUP		Total
	A	B	
MALE	52 (52%)	26 (52%)	78 (52%)
FEMALE	48 (48%)	24 (48%)	72 (48%)
Total	100 (100%)	50 (100%)	150 (100%)

P value between groups is 1.0 statistically not significant.

There was no statistically significant difference noted in the distribution of sex between the groups A & B. (P > 0.05).

DISTRIBUTION OF SMOKERS BETWEEN GROUPS A & B:

TABLE – 5:

SMOKING	GROUP		Total
	A	B	
NO	70 (70.0%)	33 (66.0%)	103 (68.7%)
YES	30 (30.0%)	17 (34.0%)	47 (31.3%)
TOTAL	100 (100.0%)	50 (100.0%)	150 (100.0%)

P value between groups is 0.709 statistically not significant.

There was no statistically significant difference noted between the groups A & B regarding smoking (P > 0.05).

DISTRIBUTION OF ALCOHOLICS BETWEEN GROUPS A & B:

TABLE – 6:

ALCOHOL	GROUP		Total
	A	B	
NO	69 (69.0%)	32 (64.0%)	101 (67.3%)
YES	31 (31.0%)	18 (36.0%)	49 (32.7%)
TOTAL	100 (100.0%)	50 (100.0%)	150 (100.0%)

P value between groups is 0.582 statistically not significant.

There was no statistically significant difference noted between the groups A & B regarding alcohol. (P > 0.05).

LIFESTYLE DISTRIBUTION BETWEEN GROUPS A & B:

TABLE – 7:

LIFESTYLE	GROUP		Total
	A	B	
SEDENTARY	52 (52.0%)	24 (48.0%)	76 (50.7%)
NON SEDENTARY	48 (48.0%)	26 (52.0%)	74 (49.3%)
TOTAL	100 (100.0%)	50 (100.0%)	150 (100.0%)

P value between groups is 0.730 statistically not significant.

There was no statistically significant difference noted in lifestyle between the groups A & B. ($P > 0.05$).

DISTRIBUTION OF FAMILY HISTORY OF HYPERTENSION BETWEEN GROUP A & GROUP B:

TABLE – 8:

FAMILY HISTORY OF HYPERTENSION	GROUP		Total
	A	B	
NO	63 (63.0%)	34 (68.0%)	97 (64.7%)
YES	37 (37.0%)	16 (32.0%)	53 (35.3%)
TOTAL	100 (100.0%)	50 (100.0%)	150 (100.0%)

P value between groups is 0.590 statistically not significant.

There was no statistically significant difference noted in the distribution of family history between the groups A & B. ($P > 0.05$).

BMI [77] DISTRIBUTION BETWEEN GROUPS A & B:

TABLE – 9:

BMI (Kg / m^2)	GROUP		Total
	A	B	
BMI < 25	51 (51.0%)	23 (46.0%)	74 (49.3%)
BMI > 25	49 (49.0%)	27 (54.0%)	76 (50.7%)
TOTAL	100 (100.0%)	50 (100.0%)	150 (100.0%)

P value between groups is 0.606 statistically not significant.

There was no statistically significant difference noted in the distribution of BMI between the groups A & B. (P > 0.05).

SYSTOLIC AND DIASTOLIC BLOOD PRESSURE DISTRIBUTION BETWEEN GROUPS A & B:

TABLE – 10:

BLOOD PRESSURE (mmHg)	GROUP	N	Mean	Std. Deviation	Std. Error Mean
SYSTOLIC	A	100	169.2800	15.81847	1.58185
	B	50	111.7200	6.88103	.97312
DIASTOLIC	A	100	101.4000	8.61054	.86105
	B	50	71.1600	6.31280	.89276

P value between groups is 0.000 < 0.001 statistically significant.

There was a statistically significant difference noted in the distribution of blood pressure (systolic & diastolic) between the groups A & B. (P < 0.05).

DISTRIBUTION OF TOTAL SERUM CALCIUM LEVELS

BETWEEN GROUP A AND GROUP B:

TABLE – 11:

TOTAL SERUM CALCIUM (mg/dl)	GROUP	N	Mean	Std.	Std. Error
	A	100	8.9160	.62529	.06253
	B	50	9.7042	.79350	.11222

P value between groups is 0.000 < 0.001 statistically significant.

There was a statistically significant difference noted in the total serum calcium levels between the groups A & B. ($P < 0.05$).

DISTRIBUTION OF CORRECTED CALCIUM LEVELS BETWEEN

GROUP A AND GROUP B:

TABLE – 12:

CORRECTED SERUM CALCIUM (mg/dl)	GROUP	N	Mean	Std.	Std. Error
	A	100	8.8263	.65250	.06525
	B	50	9.6550	.80053	.11321

P value between groups is 0.000 < 0.001 statistically significant.

There was a statistically significant difference noted in the corrected calcium levels between the groups A & B. ($P < 0.05$).

DISTRIBUTION OF SERUM PHOSPHORUS LEVELS BETWEEN GROUP A AND GROUP B:

TABLE – 13:

SERUM PHOSPHORUS (mg/dl)	GROUP	N	Mean	Std.	Std. Error
	A	100	3.6829	.39549	.03955
	B	50	3.7866	.37802	.05346

P value between groups is 0.127 not significant.

There was no statistically significant difference noted in the total serum phosphorus levels between the groups A &B($P < 0.05$).

DISTRIBUTION OF SERUM ALBUMIN LEVELS BETWEEN GROUP A AND GROUP B:

TABLE – 14:

SERUM ALBUMIN (g/dl)	GROUP	N	Mean	Std. Deviation	Std. Error Mean
	A	100	4.1169	.33042	.03304
	B	50	4.0780	.36100	.05105

P value between groups is 0.511 not significant

There was no statistically significant difference noted in the total serum albumin levels between the groups A &B($P < 0.05$).

CORRELATION BETWEEN SYSTOLIC BLOOD PRESSURE AND TOTAL SERUM CALCIUM LEVELS IN GROUP A:

TABLE – 15:

GROUP A		SYSTOLIC BP (mmHg)	TOTAL SERUM CALCIUM(mg/dl)
SYSTOLIC BP (mmHg)	Pearson Correlation	1	-.466**
	Sig. (2-tailed)		.000
	N	100	100
TOTAL SERUM CALCIUM (mg/dl)	Pearson Correlation	-.466**	1
	Sig. (2-tailed)	.000	
	N	100	100

**. Correlation is significant at the 0.01 level (2-tailed).

The correlation co-efficient was – 0.466 and p value was **0.000** which showed that the total serum calcium levels had **a significant negative correlation** with systolic blood pressure.

CORRELATION BETWEEN SYSTOLIC BLOOD PRESSURE AND CORRECTED CALCIUM LEVELS IN GROUP A:

TABLE – 16:

GROUP A		SYSTOLIC BP (mmHg)	CORRECTED SERUM CALCIUM (mg/dl)
SYSTOLIC BP (mmHg)	Pearson Correlation	1	-.526**
	Sig. (2-tailed)		.000
	N	100	100
CORRECTED SERUM CALCIUM (mg/dl)	Pearson Correlation	-.526**	1
	Sig. (2-tailed)	.000	
	N	100	100

**. Correlation is significant at the 0.01 level (2-tailed).

The correlation co-efficient was – 0.526 and p value was **0.000** which showed that the corrected serum calcium levels had **a significant negative correlation** with systolic blood pressure.

CORRELATION BETWEEN DIASTOLIC BLOOD PRESSURE AND TOTAL SERUM CALCIUM LEVELS IN GROUP A:

TABLE – 17:

GROUP A		TOTAL SERUM CALCIUM (mg/dl)	DIASTOLIC BP (mmHg)
TOTAL SERUM CALCIUM (mg/dl)	Pearson Correlation	1	-.154**
	Sig. (2-tailed)		.08
	N	100	100
DIASTOLIC BP (mmHg)	Pearson Correlation	-.154**	1
	Sig. (2-tailed)	0.08	
	N	100	100

**. Correlation is significant at the 0.01 level (2-tailed).

The correlation co-efficient was – 0.154 and p value was **0.08** which showed that the total serum calcium levels had **no significant correlation** with diastolic blood pressure.

CORRELATION BETWEEN DIASTOLIC BLOOD PRESSURE AND CORRECTED CALCIUM LEVELS IN GROUP A:

TABLE – 18:

GROUP A		DIASTOLIC BP (mmHg)	CORRECTED SERUM CALCIUM(mg/dl)
DIASTOLIC BP (mmHg)	Pearson Correlation	1	-.142**
	Sig. (2-tailed)		.102
	N	100	100
CORRECTED SERUM CALCIUM (mg/dl)	Pearson Correlation	-.142**	1
	Sig. (2-tailed)	.102	
	N	100	100

**. Correlation is significant at the 0.01 level (2-tailed).

The correlation co-efficient was – 0.142 and p value was **0.102** which showed that the corrected serum calcium levels had **no significant correlation** with diastolic blood pressure.

CORRELATION OF THE CALCIUM LEVELS WITH VARIOUS AGE GROUPS IN GROUP A

TABLE – 19:

GROUP A	Age(Yrs)	N	Mean	Std. Deviation	Std. Error	P VALUE
Total serum calcium (mg/dl)	< 40	3	9.5467	.90118	.52030	**.279**
	41 – 50	33	8.8891	.67116	.11683	
	51 – 60	32	8.8794	.54102	.09564	
	61 – 70	21	8.8114	.61852	.13497	
	>70	11	9.1309	.62815	.18939	
	Total	100	8.9160	.62529	.06253	
Corrected serum calcium (mg/dl)	< 40	3	9.1200	.91060	.52574	**.621**
	41 – 50	33	8.7564	.67147	.11689	
	51 – 60	32	8.7941	.56777	.10037	
	61 – 70	21	8.8129	.63399	.13835	
	>70	11	9.0755	.83016	.25030	
	Total	100	8.8263	.65250	.06525	

There was **no statistically significant** difference noted in the total and corrected serum calcium levels with different age groups in the study population, group A ($P > 0.05$).

CORRELATION OF CALCIUM LEVELS WITH SEX IN GROUP A:

TABLE – 20:

GROUP A	SEX	N	Mean	Std.	Std. Error	P
Total serum calcium (mg/dl)	Male	52	8.9506	.63474	.08802	**.568**
	Female	48	8.8785	.61937	.08940	
Corrected serum calcium (mg/dl)	Male	52	8.8744	.68507	.09500	**.446**
	Female	48	8.7742	.61823	.08923	

There was **no statistically significant** difference noted in the total and corrected serum calcium levels in relation to sex in group A (P > 0.05).

CORRELATION OF THE CALCIUM LEVELS WITH SMOKING IN GROUP A:

TABLE – 21:

GROUP A	SMOKING	N	Mean	Std. Deviation	Std. Error Mean	P VALUE
Total serum calcium(mg/dl)	YES	30	9.0600	.53540	.09775	**.132**
	NO	70	8.8543	.65393	.07816	
Corrected serum calcium (mg/dl)	YES	30	8.9883	.59871	.10931	**.092**
	NO	70	8.7569	.66627	.07963	

There was **no statistically significant** difference noted in the total and corrected serum calcium levels in group A in relation to smoking (P > 0.05).

CORRELATION OF THE CALCIUM LEVELS & ALCOHOL IN GROUP A:

TABLE – 22:

GROUP A	ALCOHOL	N	Mean	Std. Deviation	Std. Error Mean	P VALUE
Total serum calcium(mg/dl)	YES	31	8.9829	.61192	.10990	**.476**
	NO	69	8.8859	.63330	.07624	
Corrected serum calcium(mg/dl)	YES	31	8.9577	.64731	.11626	**.178**
	NO	69	8.7672	.65083	.07835	

There was **no statistically significant** difference noted in the total and corrected serum calcium levels in group A in relation to alcohol.
(P > 0.05).

CORRELATION OF THE CALCIUM LEVELS & LIFESTYLE IN GROUP A:

TABLE – 23:

GROUP A	LIFE STYLE	N	Mean	Std. Deviation	Std. Error Mean	P VALUE
Total serum calcium(mg/dl)	S	52	9.0233	.62265	.08635	**.074**
	NS	48	8.7998	.61347	.08855	
Corrected serum calcium(mg/dl)	S	52	8.9765	.60509	.08391	**.084**
	NS	48	8.6635	.66895	.09655	

There was **no statistically significant** difference noted in the total and corrected serum calcium levels in relation to lifestyle in group A ($P > 0.05$).

CORRELATION OF THE CALCIUM LEVELS AND FAMILY HISTORY OF HYPERTENSION IN GROUP A:

TABLE – 24:

GROUP A	FAMILY HISTORY	N	Mean	Std. Deviation	Std. Error Mean	P VALUE
Total serum calcium(mg/dl)	YES	37	8.9389	.63140	.10380	**.780**
	NO	63	8.9025	.62637	.07891	
Corrected serum calcium(mg/dl)	YES	37	8.8484	.66012	.10852	**.797**
	NO	63	8.8133	.65296	.08227	

There was **no statistically significant** difference noted in the total and corrected serum calcium levels in relation to family H/o hypertension in group A ($P > 0.05$).

CORRELATION OF THE CALCIUM LEVELS AND BMI IN GROUP A:

TABLE – 25:

GROUP A	BMI (Kg/m^2)	N	Mean	Std. Deviation	Std. Error Mean	P VALUE
Total serum calcium(mg/dl)	< 25	51	8.8453	.63747	.08926	**.251**
	> 25	49	8.9896	.61013	.08716	
Corrected serum calcium(mg/dl)	< 25	51	8.7635	.72770	.10190	**.329**
	> 25	49	8.8916	.56390	.08056	

There was **no statistically significant** difference noted in the total and corrected serum calcium levels in relation to BMI in group A ($P > 0.05$).

DISCUSSION

Systemic hypertension remains the most common, readily identifiable and reversible risk factor for myocardial infarction, stroke, heart failure, atrial fibrillation, aortic dissection and peripheral arterial disease.[2]

Evidence is growing that calcium physiology is altered in essential hypertension, but whether this is a secondary association or a causal relationship is unresolved. Intracellular calcium ions are known to have direct effects on peripheral vascular tone and it has been reported in various trials that hypertensive persons have increased concentrations of intracellular free calcium that decrease to normal levels with antihypertensive treatment.[67]

TOTAL AND CORRECTED SERUM CALCIUM LEVELS:

Various epidemiological studies stated that the calcium status of humans with essential hypertension and genetic animal models of hypertension is characterized by low serum total and ionized calcium concentration, increased intracellular calcium, increased urinary calcium excretion, and increased parathyroid hormone (PTH) concentration.[3-6,78]

This study used total serum calcium and corrected serum calcium, the latter is an alternative but not a substitute to serum ionised calcium.

In this study the mean total serum calcium and corrected serum calcium levels in group A(cases) were 8.9160 ± 0.62529 mg/dl and 8.8263 ± 0.6525 mg/dl while the mean total serum calcium and corrected serum calcium levels in group B(controls) were 9.7042± 0.79350mg/dl and 9.6550 ± 0.80053 mg/dl respectively. Statistical analysis revealed that the total and corrected serum calcium levels were significantly lowered in essential hypertensives when compared to their matched normotensive controls ($P < 0.001$ and $P < 0.001$ respectively).This observation is supported by some of the following studies.

According to K. Sudhakar et al.,[5] The mean total serum calcium levels were significantly ($p<0.01$) decreased in males and females in hypertensive group when compared with normotensive controls. In the first-degree relatives also the total serum calcium levels were significantly decreased ($p <0.01$) when compared with the controls.

AR Folsom et al.,[64] studied the serum calcium fractions in essential hypertensive and matched normotensive subjects. In their study he observed hypertensive subjects had lower mean serum levels of ultrafilterable calcium ($p = 0.01$), ionized calcium ($p = 0.09$), and complexed calcium ($p= 0.04$) and higher levels of protein-bound calcium ($p = 0.07$). Calculated serum concentrations of complexed calcium was significantly lower in

hypertensive subjects ($p = 0.04$), while proteinbound calcium concentrations was higher ($p = 0.07$). Serum phosphorus and albumin concentrations, as well as estimated dietary calcium intake, were not different between the two groups.

Fu Y, Wang S., et al.,[65] in their study suggested that that the hypertensive group consistently demonstrated a significant decreased activity of ATPase studied, with significantly lower plasma calcium and higher cytosolic calcium levels when compared with those in normotensive group ($P < 0.01$ or $P < 0.05$, respectively). No significant differences were found in either plasma Mg^{2+} or intracellular Mg^{2+} level between the two groups.

Strazzullo P et al.,[66] studied several of the biochemical abnormalities of calcium metabolism and were able to detect a significant reduction in total serum calcium levels in hypertensive subjects, although unable to detect a significant reduction in serum ionized calcium levels.This study also reported total and fractional urinary calcium excretion were elevated in subjects with essential hypertension.

Erne P, Bolli P., et al.,[67] in their study on “correlation of platelet calcium with blood pressure: effect of antihypertensive therapy” reported a decrease in the serum total calcium concentration in essential hypertensive

patients. Touyz, R.M.,et al., [4] also reported a decrease in the serum total calcium concentration in essential hypertensive patients.

Some investigators; McCarron DA., [68] and Resnick LM, Laragh JH.,et al.,[69] also noted that, compared with normotensive subjects,essential hypertensive subjects had lower serum ionized calcium concentrations even when total calcium levels were similar.

Wright GL, Rankin GO.,[79] in their study on concentrations of ionic and total calcium in plasma of four models of hypertension noted a lower serum ionized and total serum calcium concentrations in spontaneously hypertensive rats(SHR).

Several other investigators have reported positive associations between blood pressure levels and concentrations of serum total calcium which is in contrast with the present study. Harlan WR., et al., [80] , Rolf Jorde.,et al., [81] and Kesteloot H.,et al., [82] observed a direct correlation between serum total calcium concentrations and arterial pressure. This seemingly paradoxical observation is likely secondary to a failure of the investigators to correct for the hemoconcentration that attends human hypertension.[83] This failure results in an artifactual increase in total calcium values because of an increase in serum protein (albumin) concentration, the fraction to which most calcium is bound.

CORRELATION OF TOTAL AND CORRECTED SERUM CALCIUM LEVELS WITH SYSTOLIC BLOOD PRESSURE:

In this present study a correlation between calcium levels and systolic blood pressure was attempted and found that the total and corrected serum calcium levels had a significant negative correlation with systolic blood pressure ($P < 0.01$ and $P < 0.01$ respectively).

Ottar Hals [84] in his study found that pretreatment systolic blood pressure was inversely correlated to serum ionized calcium ($r = — 0.44$, and $p = 0.05$). The result in this study supports our study. Also according to Morris, C.D.et al., [84] and Christina Martinez [85], there was a clear inverse relationship between calcium and both the prevalence of hypertension and the level of blood pressure.

AR Folsom et al., [64] studied the serum calcium fractions in essential hypertensive and matched normotensive subjects. In his study, there was no significant correlation between individual systolic blood pressure levels and the serum calcium fractions.

Phillips AN.,et al., [87] in their "The British Regional Heart Study" found a significant positive correlation between serum calcium and both systolic and diastolic blood pressure after adjusting for age. This association

was diminished after adjustment for serum albumin, but remained significant. This association disappeared after adjustment for serum globulins and haematocrit in addition to age and serum albumin. Thus, these factors appear to mediate the weak association between serum calcium and both systolic and diastolic blood pressure. There does not appear to be an independent relationship between serum calcium and blood pressure.

Some works done by Staessen J, Sartor F,et al., [88] Jorde.,et al., [81] and Kesteloot H.,et al., [82] showed that serum total calcium was independently and positively correlated with systolic pressure in essential hypertensives.

CORRELATION OF TOTAL AND CORRECTED SERUM CALCIUM LEVELS WITH DIASTOLIC BLOOD PRESSURE:

In our study we also attempted a correlation between the calcium levels and diastolic blood pressure and found that there was no correlation between the total and corrected serum calcium levels and diastolic blood pressure ($P > 0.05$ and $P > 0.05$ respectively).

In his study on 'the serum calcium fractions in essential hypertensive and matched normotensive subjects' AR Folsom et al.,[64] also noticed that there was no significant correlations between the serum calcium fractions and diastolic blood pressure levels.

Jorde.,et al., [81]and Kesteloot H, Geboers J.,[82] showed a significant positive correlation between total serum calcium and diastolic blood pressure. Kesteloot H, Joossens JV.,[89] showed that serum calcium correlated positively with systolic and diastolic blood pressure in men, but only with diastolic blood pressure in women.

CORRELATION OF TOTAL AND CORRECTED SERUM CALCIUM LEVELS WITH VARIOUS SUBSETS OF STUDY GROUP(ESSENTIAL HYPERTENSIVE GROUP):

Our study also had an objective of comparing total and corrected serum calcium levels with various subsets of essential hypertensive population like age, sex, smoking, alcohol, family history of hypertension, lifestyle and BMI. After statistical analysis, it was revealed that there was no significant difference between the calcium levels in the above mentioned parameters($P > 0.05$).

Jorde.,et al., [81] , in his study noticed that there was a significant decrease in serum calcium with increasing age in men while a significant increase in women. According to AR Folsom et al.,[64] and Staessen J, Sartor F,et al., [88] serum total calcium was similar in both the sexes and no significant difference noted.

According to C Brot.,et al., [90] noted that there was no difference in serum ionized calcium between smokers and non-smokers in their study. J. Sundsfjord., R. Jorde., et al., [91] in an observation of tromso study, showed a positive association of serum calcium with body mass index (BMI) in both sexes that persisted after correcting for other variables in a multiple regression model. Physical activity(lifestyle) had no significant association with serum calcium.In females alcohol consumption was negatively, and cigarette smoking was positively associated with serum calcium.

K. Sudhakar et al.,[5] found that the first-degree relatives of essential hypertensive patients had a significantly decreased ($p<0.01$) total serum calcium levels when compared with the controls.

LIMITATIONS OF THE STUDY:

1. The sample size was small.
2. This being a cross sectional study follow up was not done.
3. Serum ionised calcium, cytosolic calcium, urinary calcium, serum parathormone levels, serum renin levels, and serum Vit D_3 levels were not measured due to constraints.
4. Calcium supplementation was not attempted in the patients due to ethical reasons.

SUMMARY

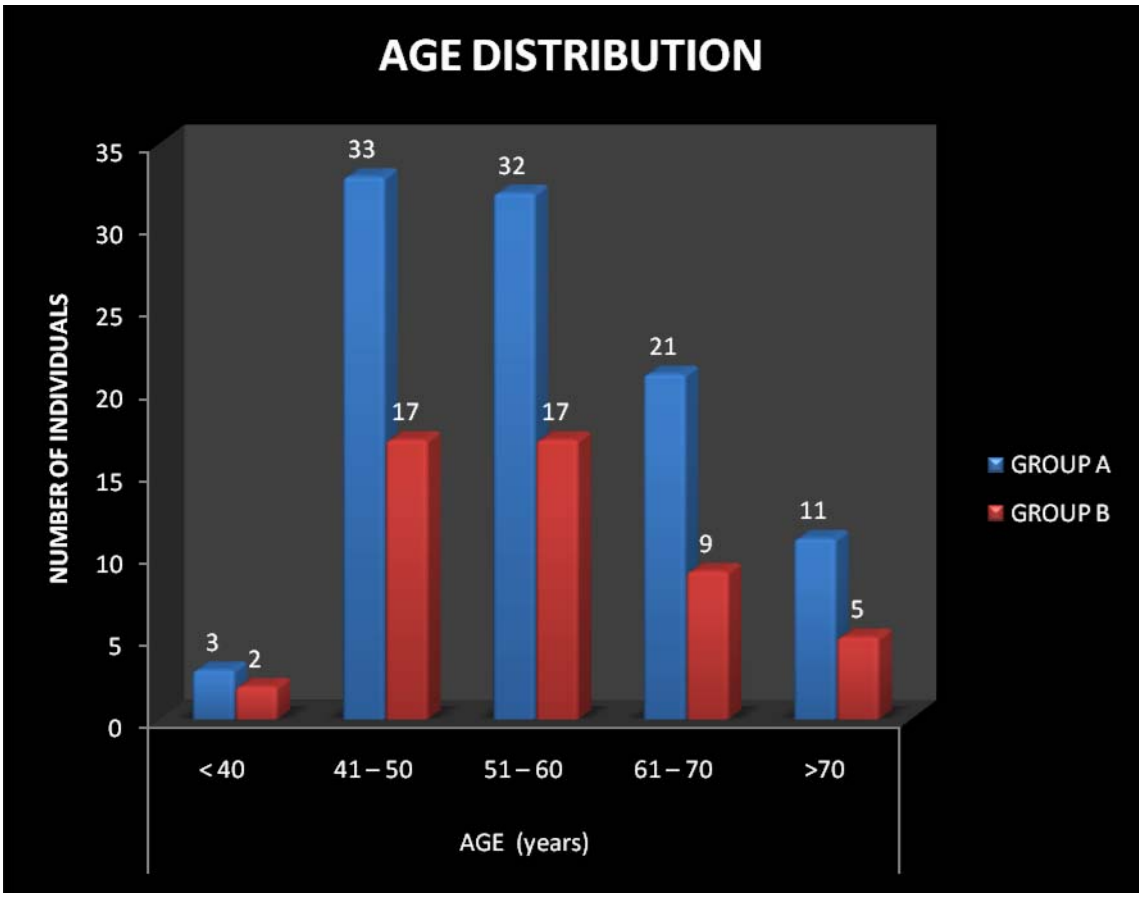
The present study aimed at comparing the total and corrected serum calcium levels in newly detected essential hypertensive patients with matched normotensive controls. It also aimed at correlating the total and corrected serum calcium levels with systolic and diastolic blood pressure in newly detected essential hypertensive patients. With rigid criteria 150 individuals, 100 essential hypertensives cases and 50 normotensive controls were selected and enrolled in the study.

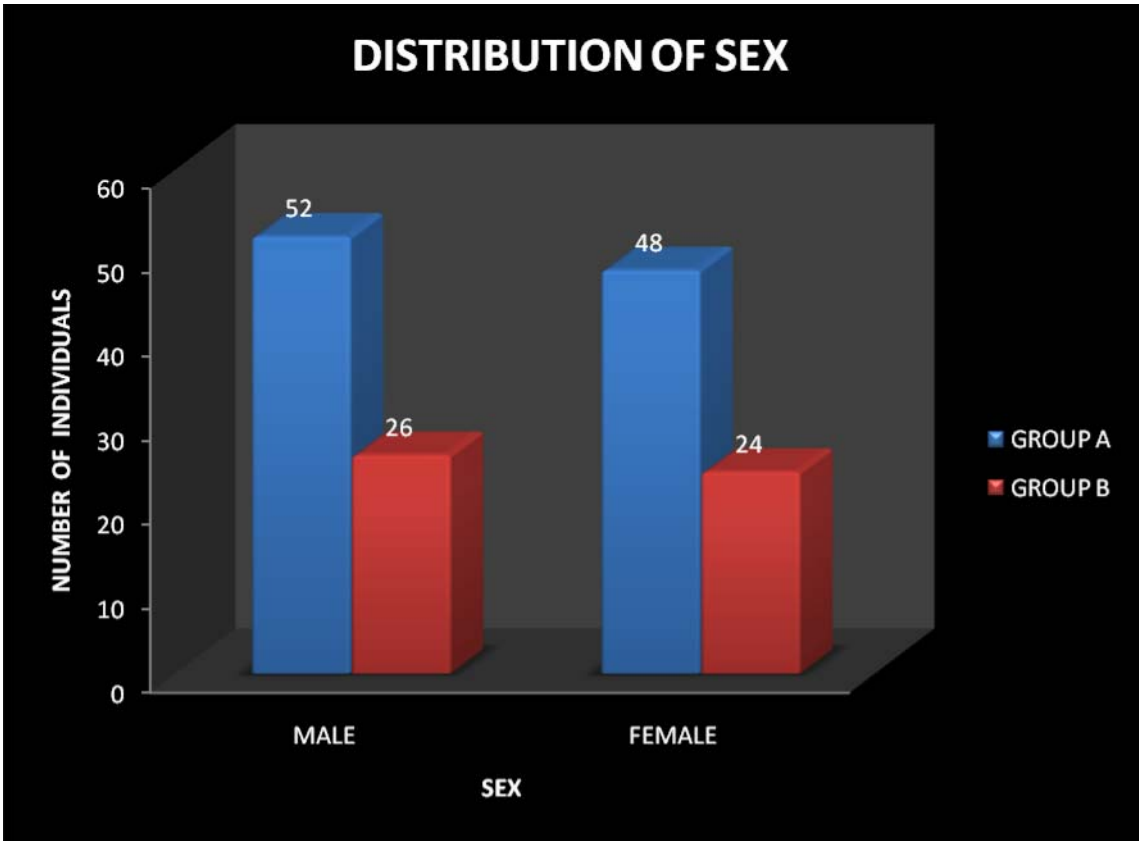
The total and corrected serum calcium levels were found to be significantly lowered in cases when compared to controls. Also a significant negative correlation between the calcium levels and systolic blood pressure was noted while there was no correlation noted with the diastolic blood pressure in the cases. This study also noted that there is no significant difference in both the calcium levels with age, sex, BMI, lifestyle, smoking, alcohol, family H/o hypertension in newly detected essential hypertensive patients.

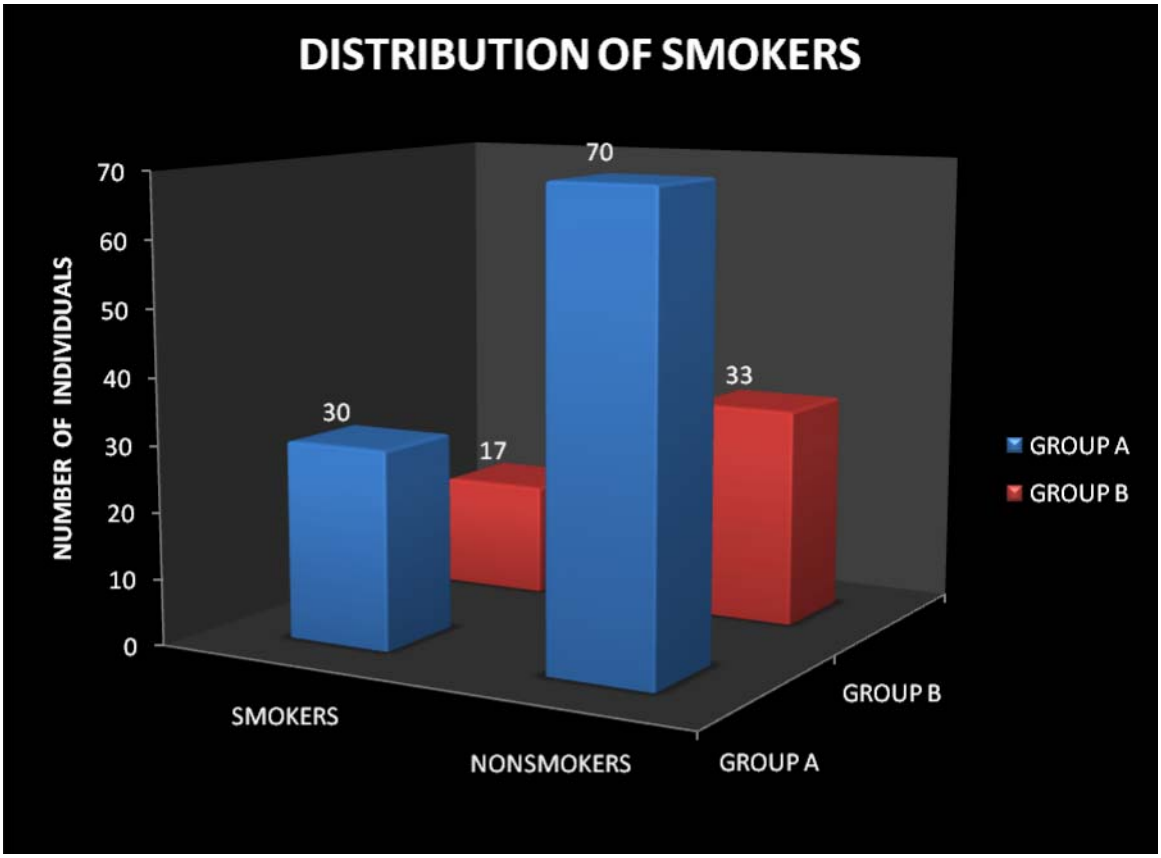
This study recommends calcium intake in essential hypertensives mainly in the form of fruits and non-fatty dairy products similar to DASH trials. As calcium supplements may rarely raise blood pressure and increase the risk of kidney stones, further larger studies with randomised controlled trials are necessary to support the view on calcium supplementation.

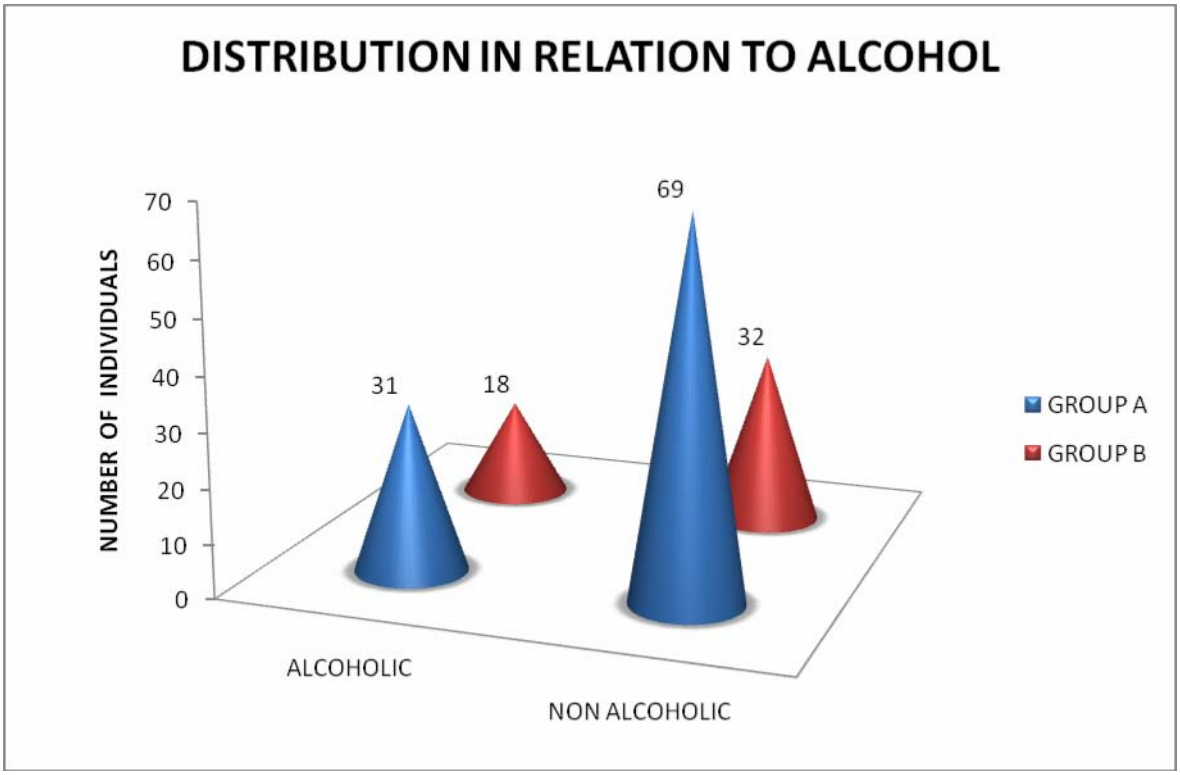
CONCLUSION

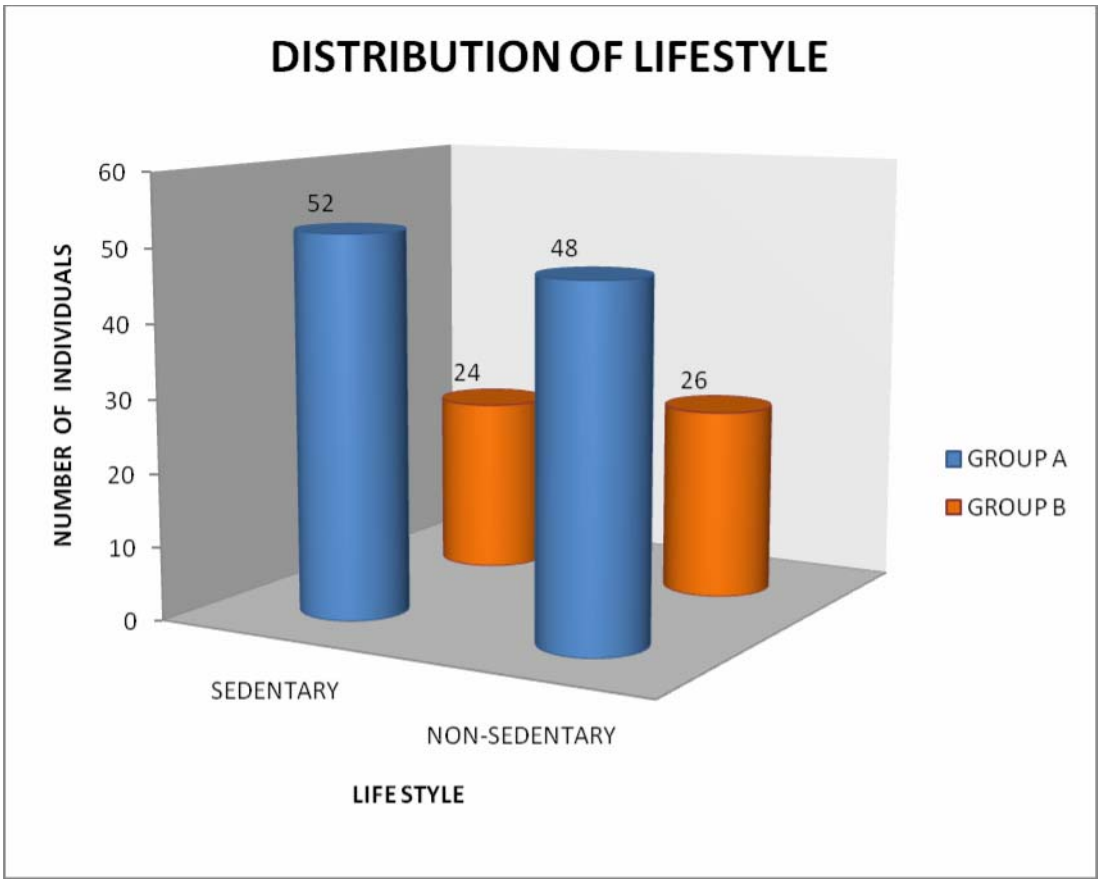
1. The total and corrected serum calcium levels are significantly lowered in newly detected essential hypertensive patients when compared to normotensive controls.
2. The total and corrected serum calcium levels have a significant negative correlation with the level of systolic blood pressure in newly detected essential hypertensive patients.
3. The total and corrected serum calcium levels have no significant correlation with the diastolic blood pressure in newly detected essential hypertensive patients.
4. The total and corrected serum calcium levels showed no significant difference with age , sex , BMI , life style , smoking , alcohol ,family history of hypertension in newly detected essential hypertensive patients.

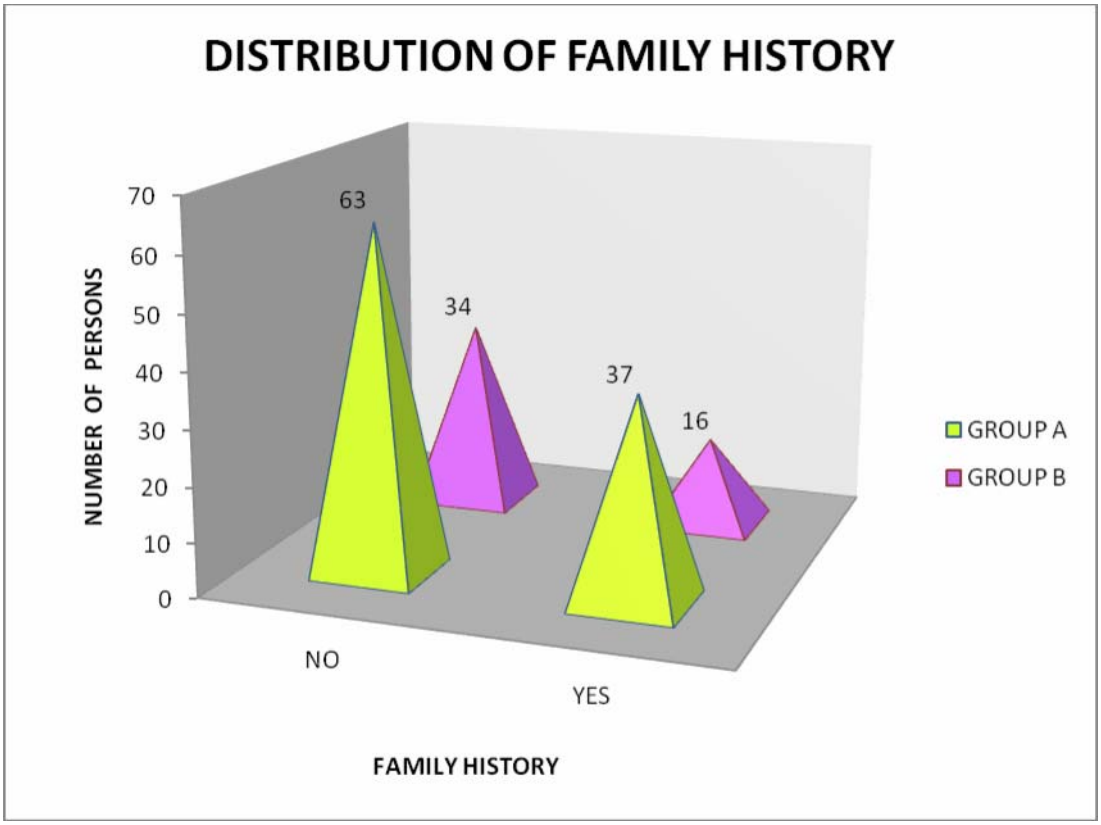

AGE DISTRIBUTION
NUMBER OF INDIVIDUALS
35
30
25
20
15
10
5
0
33
32
21
17
17
11
9
5
3
2
<40
41 – 50
51 – 60
61 – 70
>70
AGE (years)
GROUP A
GROUP B

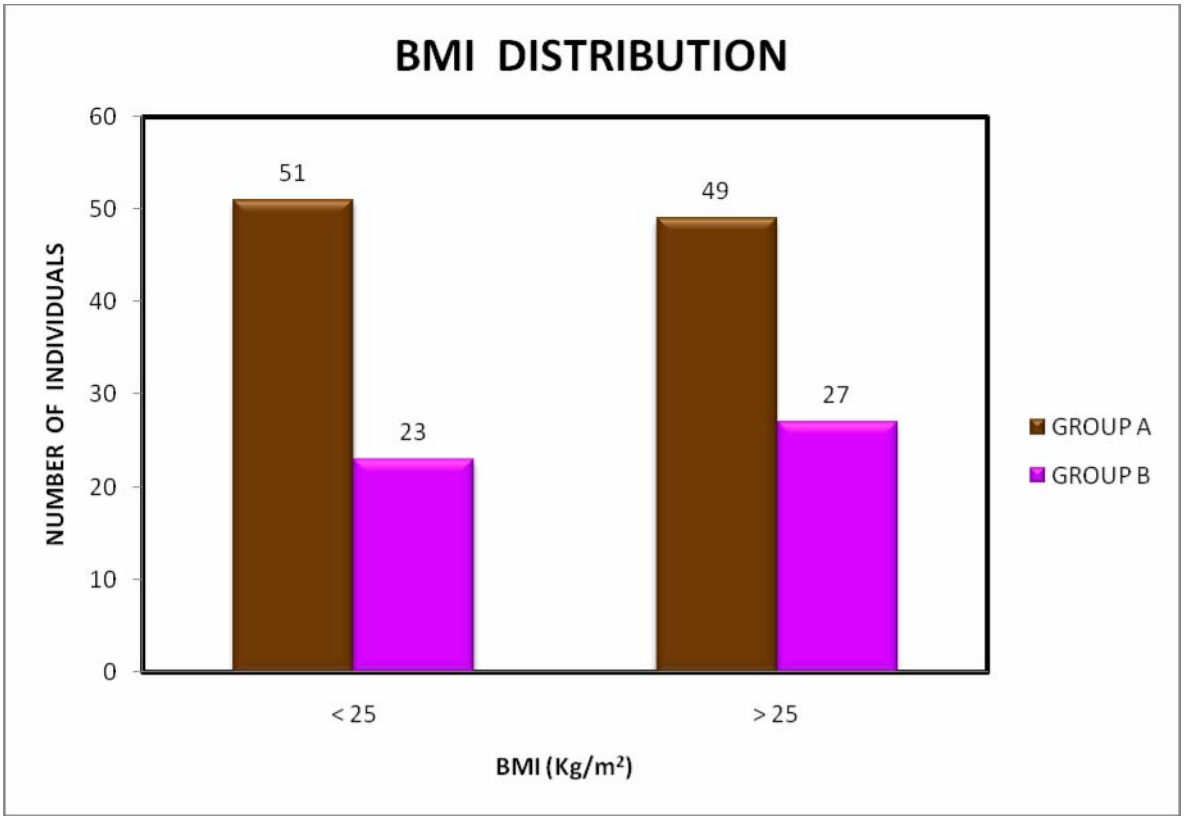

DISTRIBUTION OF SEX
NUMBER OF INDIVIDUALS
60
50
40
30
20
10
0
52
48
26
24
MALE
FEMALE
SEX
GROUP A
GROUP B

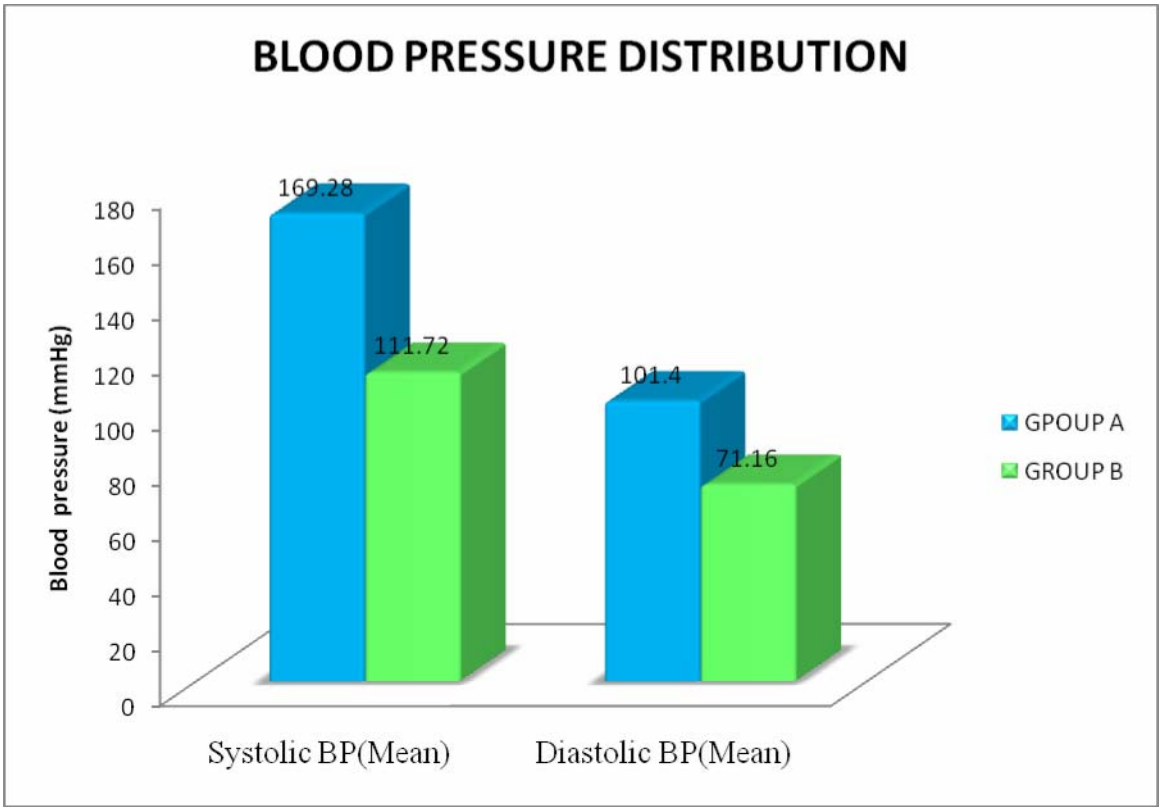

DISTRIBUTION OF SMOKERS
NUMBER OF INDIVIDUALS
70
60
50
40
30
20
10
0
30
17
70
33
SMOKERS
NONSMOKERS
GROUP A
GROUP B
GROUP A
GROUP B

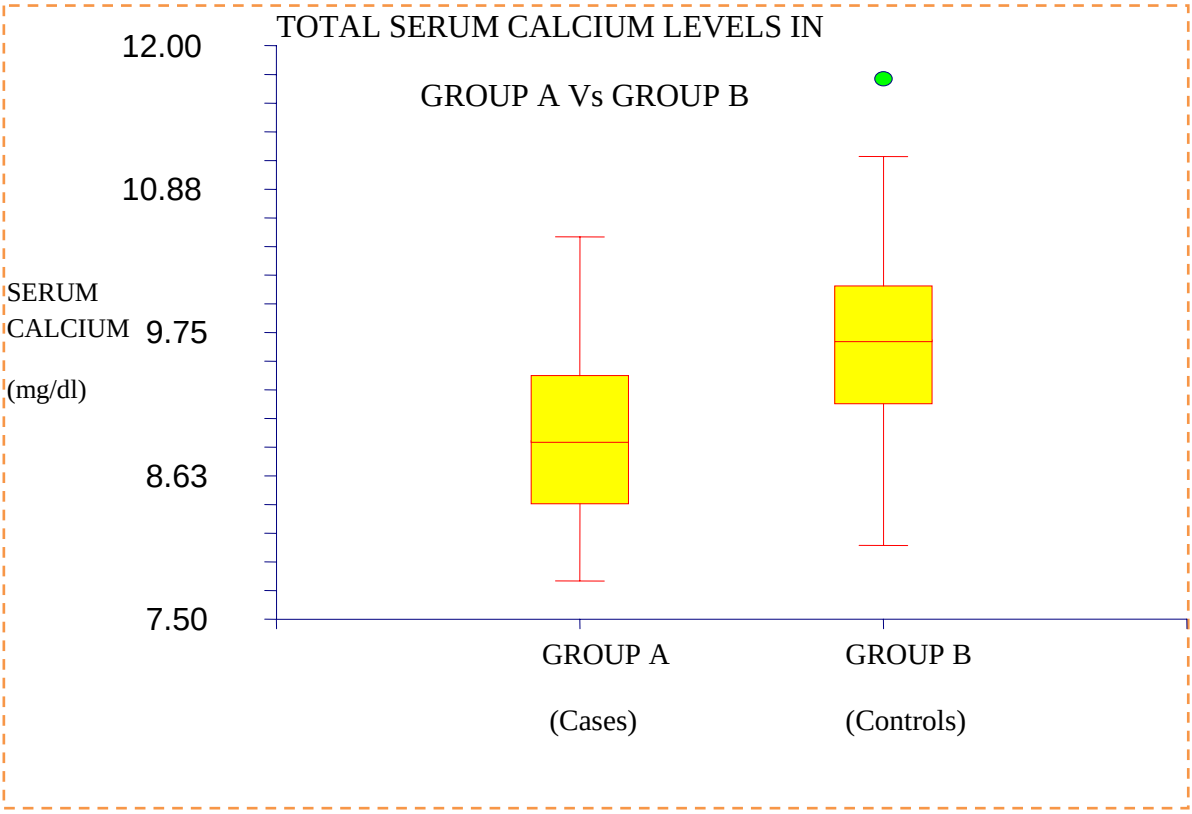

DISTRIBUTION IN RELATION TO ALCOHOL
NUMBER OF INDIVIDUALS
70
60
50
40
30
20
10
0
31
18
69
32
ALCOHOLIC
NON ALCOHOLIC
GROUP A
GROUP B

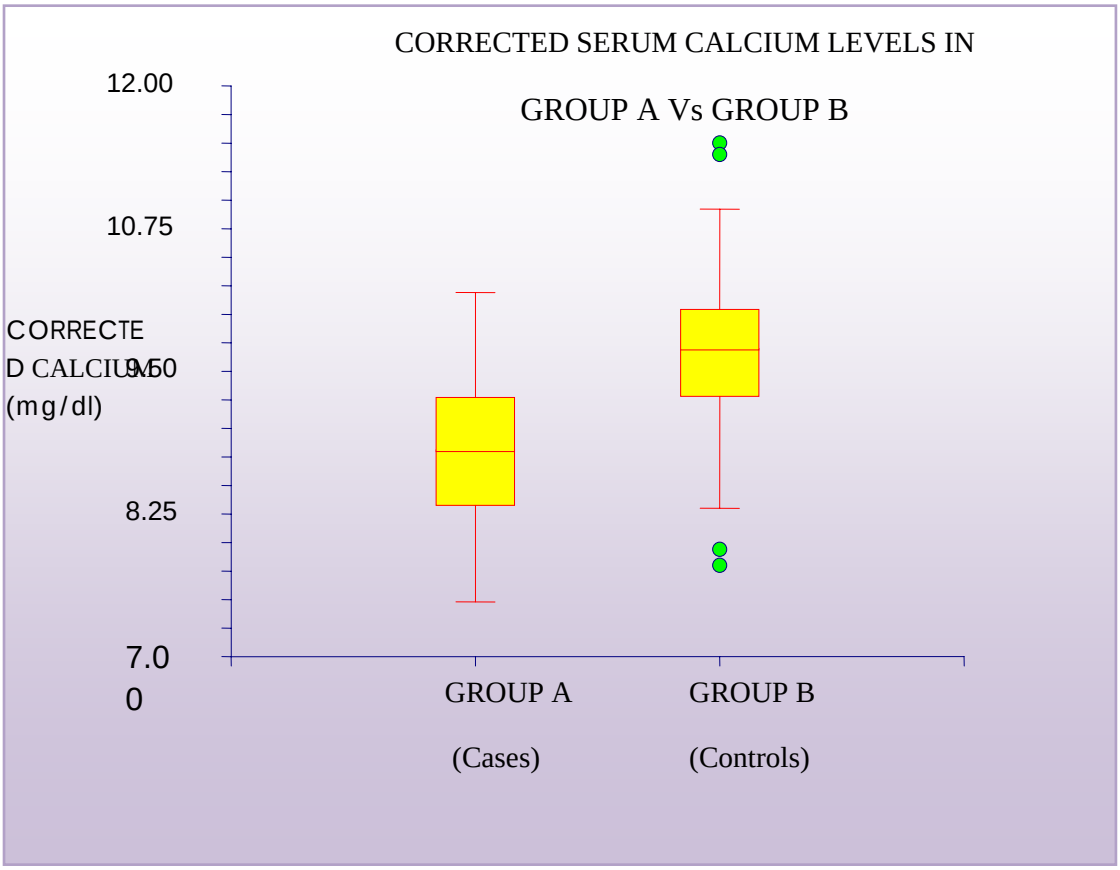

DISTRIBUTION OF LIFESTYLE
NUMBER OF INDIVIDUALS
60
50
40
30
20
10
0
52
24
48
26
SEDENTARY
NON-SEDENTARY
LIFE STYLE
GROUP A
GROUP B

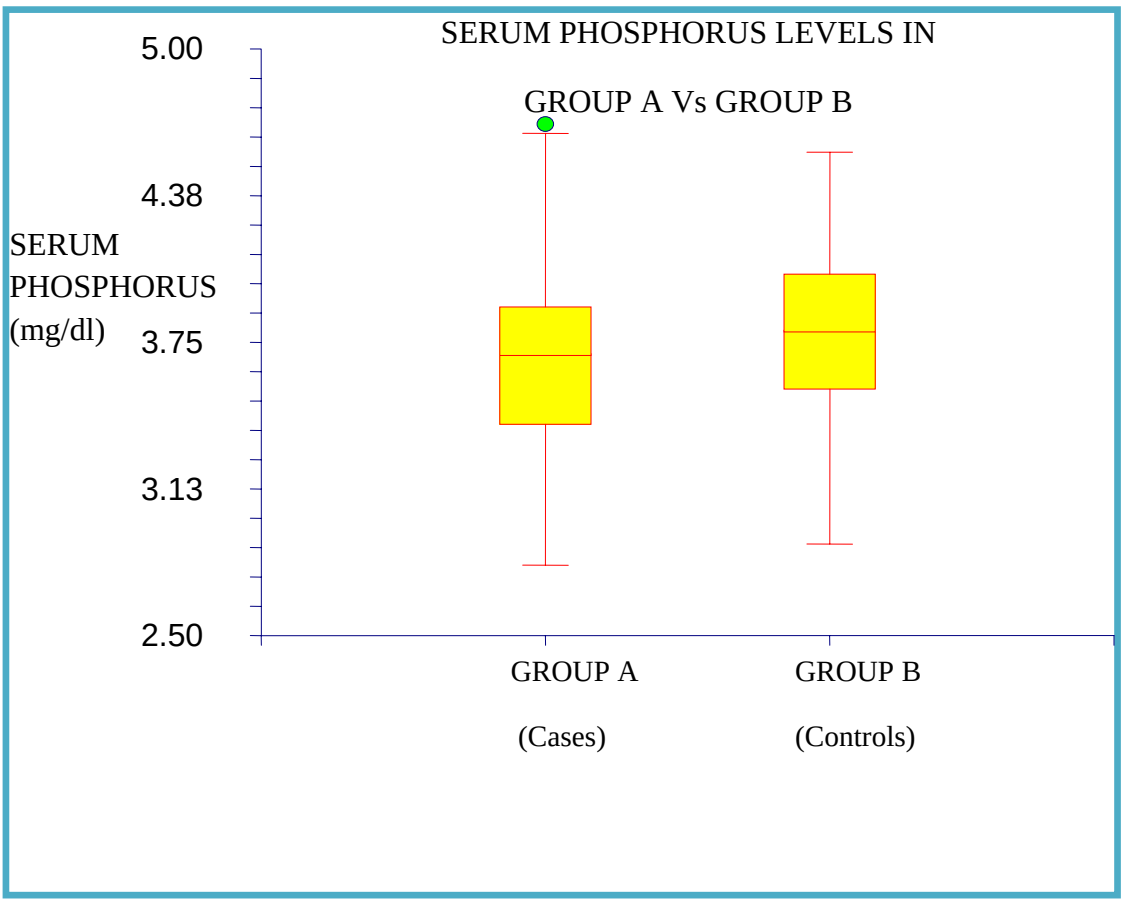

DISTRIBUTION OF FAMILY HISTORY
NUMBER OF PERSONS
70
60
50
40
30
20
10
0
63
34
37
16
NO
YES
FAMILY HISTORY
GROUP A
GROUP B

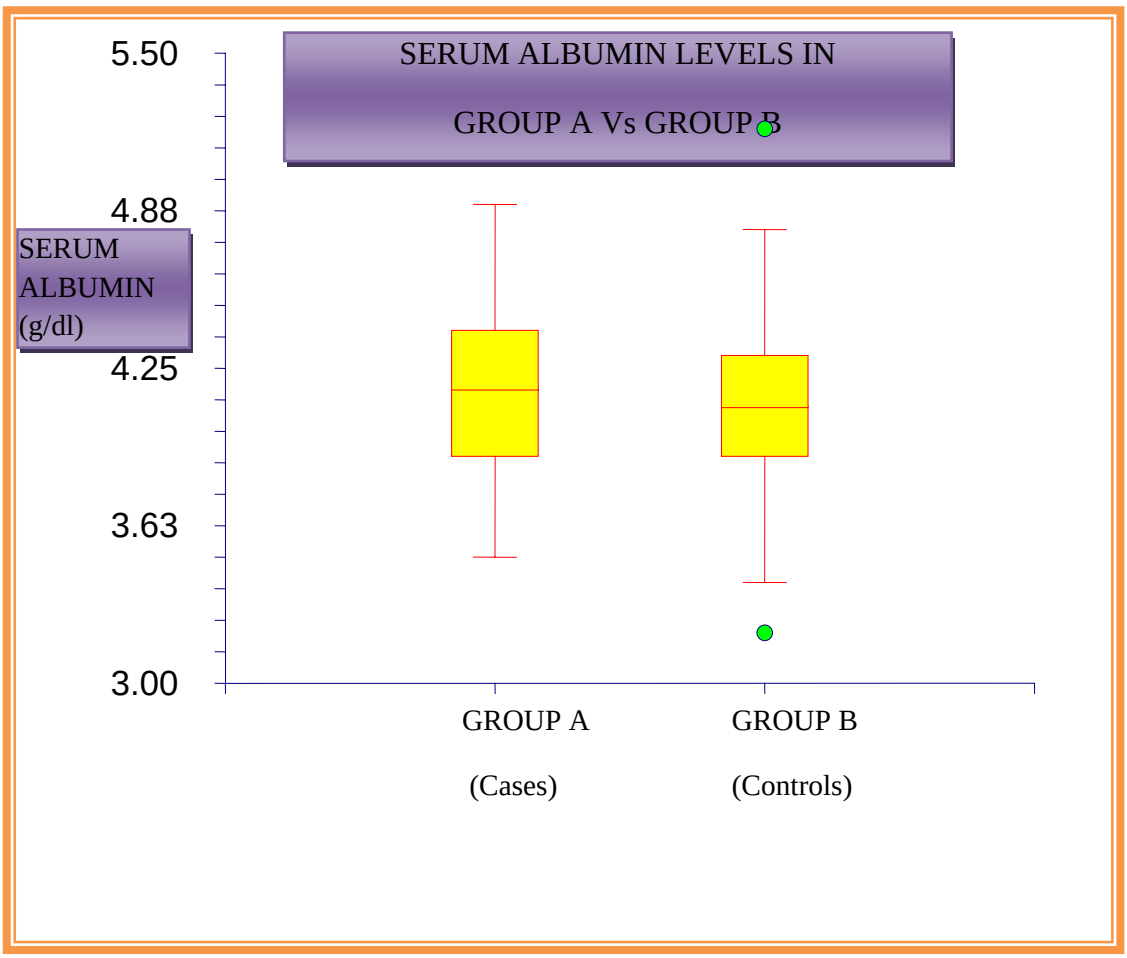

BMI DISTRIBUTION
NUMBER OF INDIVIDUALS
60
50
40
30
20
10
0
51
23
49
27
< 25
> 25
BMI (Kg/m²)
GROUP A
GROUP B


BLOOD PRESSURE DISTRIBUTION
Blood pressure (mmHg)
180
160
140
120
100
80
60
40
20
0
169.28
111.72
101.4
71.16
Systolic BP(Mean)
Diastolic BP(Mean)
GPOUP A
GROUP B


TOTAL SERUM CALCIUM LEVELS IN
GROUP A Vs GROUP B
12.00
10.88
9.75
8.63
7.50
SERUM CALCIUM (mg/dl)
GROUP A
(Cases)
GROUP B
(Controls)


CORRECTED SERUM CALCIUM LEVELS IN
GROUP A Vs GROUP B
12.00
10.75
9.50
8.25
7.00
CORRECTED CALCIUM (mg/dl)
GROUP A
(Cases)
GROUP B
(Controls)


SERUM PHOSPHORUS LEVELS IN
GROUP A Vs GROUP B
5.00
4.38
SERUM
PHOSPHORUS
(mg/dl)
3.75
3.13
2.50
GROUP A
(Cases)
GROUP B
(Controls)


SERUM ALBUMIN LEVELS IN
GROUP A Vs GROUP B
SERUM ALBUMIN (g/dl)
5.50
4.88
4.25
3.63
3.00
GROUP A
(Cases)
GROUP B
(Controls)

DEPARTMENT OF MEDICINE

Govt Kilpauk Medical College & Hospital, Chennai – 10.

PROFORMA

ESTIMATION OF SERUM CALCIUM LEVELS IN NEWLY DETECTED ESSENTIAL HYPERTENSION PATIENTS

S.No. Reg. No. (OP/IP)

Name: Age/Sex

Address:

Occupation/Income:

Presenting Complaints:

Headache	Vomiting	Giddiness
Breathlessness	Oliguria	Palpitation
Facial puffiness	Swollen Limb	Epistaxis
Syncope	Chest pain	Anorexia
Easy fatigability	Blurring of vision	Hiccough
Polyuria	Nocturia	Polydipsia
Muscle spasms	Convulsions	Paresthesia
Abdominal pain	Sweating	Tremors
Impotence	Snoring	Constipation

Weight Gain/Weight Loss Cold Intolerance

Change in Voice Menstrual Irregularities

Emotional disturbances Others

PAST HISTORY

SHT DM Angina/MI/IHD/CCF

STROKE/TIA PVD RENAL DISORDERS

BLOOD TRANSFUSION THYROID SURGERY Others

FAMILY HISTORY

HYPERTENSION (Father/Mother/Siblings) DM IHD

PERSONAL HISTORY

DIET : Veg/Non-veg LIFE STYLE: Sedentary/Non-sedentary

Addiction: Tobacco Chewing/Smoking /Drug Abuse

Alcohol-Occasional/Daily/Moderate/Heavy

Menstrual History

H/o Drug intake

GENERAL EXAMINATION

HEIGHT(m): WEIGHT(Kg): BMI(Kg/m^2) :

Pallor Cyanosis Clubbing Icterus

Pedal edema GLA JVP

Markers of Atherosclerosis

Features of Hypothyroidism/Acromegaly/Cushing's syndrome

FUNDUS EXAMINATION:

VITAL SIGNS

PR : RR : Temperature (F):

Blood pressure: RUL: I - II -

(mmHg) LUL: I - II -

SYSTEMIC EXAMINATION:

C. V. S: S1- S2- Murmur:

R. S. : NVBS Added sounds:

P/A : Organomegaly Renal bruit/ Renal angle tenderness

C. N. S:

INVESTIGATIONS:

URINE R/E: Albumin- Sugar- Deposits-

CBC : Hb (g/dl)- TLC/mm^3- DC-

ESR(mm) -

Urine Spot PCR:

Protein(mg%) - Creatine(mg%) - Ratio:

BLOOD SUGAR (mg/dl) :

BLOOD UREA (mg/dl) :

SERUM CREATININE (mg/dl) :

SERUM SODIUM (mEq/L) :

SERUM POTASSIUM (mEq/L) :

SERUM CALCIUM (mg/dl) :

SERUM PHOSPHORUS (mg/dl) :

SERUM ALBUMIN (g/dl) :

CORRECTED CALCIUM LEVEL (mg/dl) :

CHEST X-RAY: **ELECTROCARDIOGRAM:**

ABDOMINAL ULTRASONOGRAM:

MASTER CHART

ESSENTIAL HYPERTENSION PATIENTS (STUDY GROUP/ GROUP - A)

S.No	NAME	Op/Ip No	Age	Sex	Symptoms	Smoking	Alcohol	Life style	Family H/O	BMI (Kg/m^2)	BMI Group	BLOOD PRESSURE(mmHg)		Blood Urea (mg/dl)	Serum Creatinine (mg/dl)	USG Abd	T. Sr. Calcium (mg/dl)	Serum Phos (mg/dl)	Serum Albumin (g/dl)	C Sr. Calcium (mg/dl)
												Systolic	Diastolic							
1	Mr.Eesan	316	52	M	H	Y	N	S	N	26.88	2	150	94	25	0.8	N	9.26	2.8	4.2	9.1
2	Mrs.Lakshmi	333	48	F	H , G	N	N	NS	Y	27.94	2	144	96	24	0.4	N	9.38	3.6	4.4	9.06
3	Mrs.Chakkamma	342	62	F	-	N	N	NS	N	21.8	1	184	96	27	0.7	N	8.02	3.2	4.2	7.86
4	Mr.Purusothaman	352	45	M	-	Y	N	S	N	22.79	1	156	94	24	0.6	F	9.42	3.2	3.8	9.58
5	Mrs.Ellammal	405	45	F	H	N	N	NS	N	21.09	1	182	98	26	0.6	N	9	3	4.5	8.6
6	Mrs.Pattammal	412	65	F	G	N	N	S	N	20.3	1	164	102	27	0.7	N	8.15	3.6	3.5	8.55
7	Mr.Ramalingam	414	53	M	-	Y	N	S	N	20.76	1	148	90	36	1.1	N	9.7	4.1	4.4	9.38
8	Mrs.Nagammal	506	56	F	-	N	N	S	Y	27.57	2	170	106	19	1	F	8.9	4	3.8	9.06
9	Mr.Ramakrishnan	513	43	M	-	Y	N	NS	N	21.99	1	164	106	27	0.8	N	8.32	3.2	4	8.32
10	Mr.Mohamed Safi	520	54	M	-	N	N	NS	N	27.95	2	162	90	26	0.6	N	8.24	3.6	4	8.24
11	Mr.Rajan	522	52	M	-	N	Y	S	Y	20.31	1	194	114	28	0.8	N	8.42	3.5	4.5	8.02
12	Mr.Narasaiah	585	74	M	EF	N	Y	S	N	20.56	1	182	112	32	1	N	8.12	3.9	4.8	7.48
13	Mr.Parthasarathy	586	65	M	G	Y	Y	NS	N	23.18	1	158	96	28	0.8	N	9.05	3.5	4.4	8.73
14	Mrs.Suseela	636	56	F	G	N	N	NS	Y	19.62	1	186	102	24	0.6	N	8.3	4.2	4.2	8.46
15	Mr.C.Pandurangan	639	67	M	-	Y	N	NS	N	30.14	2	186	104	22	0.7	N	8.06	3.8	4.2	7.9
16	Mr.Navaneethan	642	45	M	H	N	Y	NS	Y	28.33	2	160	98	25	0.5	N	8.3	3.6	3.8	8.46
17	Mrs.Shanthi	644	58	F	-	N	N	NS	Y	27.12	2	174	108	20	0.8	N	9	3.9	4.5	8.6
18	Mrs.Thilagavathy	654	54	F	-	N	N	S	N	20.58	1	182	96	32	0.8	F	9.48	3.8	4.3	9.24
19	Mrs.Manohari	663	45	F	H	N	N	NS	N	19.06	1	178	102	22	0.6	N	8.52	2.9	4	8.52
20	Mrs.Regina	664	46	F	G	N	N	S	N	20.8	1	160	110	27	0.7	N	9.14	3.7	4.1	9.06
21	Mrs.Rajammal	665	75	F	G	N	N	NS	N	23.28	1	172	92	32	0.7	N	10	3.8	3.8	10.12
22	Mrs.Devaki	668	55	F	G	N	N	S	N	26.77	2	174	102	24	0.6	N	8.56	3.7	4.2	8.4
23	Mr.Lenin	669	55	M	-	Y	Y	S	N	20.21	1	140	92	24	0.6	N	8.5	3.5	3.5	8.9
24	Mrs.Kaliyammal	674	56	F	G	N	N	NS	N	21.05	1	166	96	27	0.9	N	8.36	4	4.4	8.04
25	Mrs.Geetha	682	41	F	-	N	N	NS	N	19.6	1	176	102	26	0.6	N	8.44	3.4	4.4	8.12

MASTER CHART

ESSENTIAL HYPERTENSION PATIENTS (STUDY GROUP/ GROUP - A)

S.No	NAME	Op/Ip No	Age	Sex	Symptoms	Smoking	Alcohol	Life style	Family H/O	BMI (Kg/m^2)	BMI Group	BLOOD PRESSURE(mmHg)		Blood Urea (mg/dl)	Serum Creatinine (mg/dl)	USG Abd	T. Sr. Calcium (mg/dl)	Serum Phos (mg/dl)	Serum Albumin (g/dl)	C Sr. Calcium (mg/dl)
												Systolic	Diastolic							
26	Mr.Meeran	688	49	M	-	Y	Y	NS	Y	20.92	1	168	98	27	0.6	N	8.8	3.9	4.1	8.72
27	Mrs.Kumutha	689	64	F	-	N	N	NS	Y	20.3	1	196	124	32	0.8	N	8.1	3.9	4.2	7.94
28	Mr.Sriram	694	73	M	EF	N	N	NS	N	21.27	1	152	98	26	0.8	N	8.32	3.6	4.2	8.16
28	Mr.Kathiresan	704	59	M	-	Y	N	NS	Y	22.22	1	172	106	22	0.7	N	8.72	2.9	4.5	8.32
30	Mrs.Parimala	826	55	F	-	N	N	S	N	25.36	2	152	98	26	0.9	N	9.02	2.9	3.9	9.1
31	Mrs.Mala	827	48	F	G , H	N	N	S	N	27.96	2	172	100	26	0.5	F	9	2.9	3.7	9.24
32	Mrs.Jamunamma	828	45	F	-	N	N	S	N	28.13	2	176	102	27	0.8	N	8.32	4	4.2	8.16
33	Mr.Ajesh	829	57	M	-	Y	Y	S	Y	26.77	2	150	92	27	0.6	N	8.5	4.1	3.5	8.9
34	Mrs.Viruthambal	830	53	F	-	N	N	S	N	28.12	2	172	98	18	0.7	N	8.66	3.6	4	8.66
35	Mr.Lingam	839	51	M	H , G	Y	N	NS	N	21.32	1	154	92	29	0.9	N	9.1	3.7	4	9.1
36	Mrs.Annammal	840	65	F	G	N	N	S	Y	26.71	2	148	94	18	0.6	N	9.29	4.2	3.5	9.69
37	Mrs.Alamelu	849	65	F	-	N	N	NS	N	32.09	2	194	116	27	0.8	N	8.37	3.7	4.1	8.29
38	Mrs.Sarojamma	866	72	F	-	N	N	NS	Y	26.95	2	174	90	28	0.9	N	8.66	3.1	4	8.66
39	Mrs.Mumtaj	870	64	F	-	N	N	S	N	27.82	2	162	100	36	1.1	N	8.5	3.9	3.8	8.66
40	Mr.Ravi	877	48	M	-	N	Y	S	N	26.18	2	170	90	18	0.9	N	8.45	3.4	4	8.45
41	Mr.R.Madavan	878	58	M	-	Y	Y	NS	Y	21.01	1	170	96	22	0.6	N	9.21	3.9	4.2	9.05
42	Mr.Manjunathan	884	43	M	H , S	N	Y	S	Y	28.12	2	156	98	34	0.9	N	8.66	3.8	3.7	8.9
43	Mr.Masilamani	885	45	M	G , H	Y	Y	S	Y	19.35	1	162	96	29	0.6	N	8.5	3.6	3.8	8.66
44	Mr.C.Ramanujam	888	55	M	G	N	Y	NS	N	27.33	2	174	108	18	0.6	N	8.24	3.3	3.9	8.32
45	Mr.Magudeswaran	890	76	M	H	Y	N	S	Y	28.67	2	150	90	28	0.8	N	9.44	3.2	3.8	9.6
46	Mr.Loganathan	917	65	M	-	N	Y	NS	Y	26.24	2	164	92	27	0.7	N	8.98	3.6	4	8.98
47	Mrs. Suguna	919	52	F	H , G	N	N	S	N	27.69	2	170	118	24	0.8	N	8.26	4.1	4.2	8.1
48	Mrs.Therasa	921	58	F	-	N	N	NS	Y	26.87	2	162	92	26	0.8	F	9	3.4	3.7	9.24
49	Mrs.Babyamma	940	65	F	G	N	N	S	N	30.45	2	182	96	20	0.7	N	8.06	3.6	3.6	8.38
50	Mrs.Malini	945	48	F	H	N	N	S	N	21.87	1	206	126	27	0.7	N	8.06	3.2	4.5	7.76

MASTER CHART

ESSENTIAL HYPERTENSION PATIENTS (STUDY GROUP/ GROUP - A)

S.No	NAME	Op/Ip No	Age	Sex	Symptoms	Smoking	Alcohol	Life style	Family H/O	BMI (Kg/m^2)	BMI Group	BLOOD PRESSURE(mmHg)		Blood Urea (mg/dl)	Serum Creatinine (mg/dl)	USG Abd	T. Sr. Calcium (mg/dl)	Serum Phos (mg/dl)	Serum Albumin (g/dl)	C Sr. Calcium (mg/dl)
												Systolic	Diastolic							
51	Mr.Deivasigamani	950	68	M	N	N	N	NS	Y	21.98	1	190	112	22	0.7	N	8.02	3.9	4.1	7.94
52	Mr.Babu	953	43	M	-	N	N	NS	N	20.07	1	186	116	17	0.7	N	7.82	3.8	4.2	7.66
53	Mrs.Kasthuri	957	43	F	H	N	N	S	N	20.95	1	174	98	32	1	N	8.5	3.7	4.2	8.34
54	Mr.S.Rajendran	960	61	M	H	Y	Y	NS	Y	21.12	1	160	98	28	0.7	N	9.72	3.6	3.5	10.12
55	Mr.Jeyakumar	969	45	M	-	N	Y	S	Y	19.14	1	174	98	26	0.8	N	8.4	4	3.8	8.56
56	Mrs.Saradha	978	70	F	G ,H	N	N	NS	N	20.31	1	164	96	33	0.8	N	9.19	3.8	3.9	9.27
57	Mrs.Kuppu	988	45	F	G	N	N	S	N	19.38	1	174	106	29	1.1	N	8.51	3.3	4.1	8.43
58	Mrs.Visalakshi	990	72	F	G	N	N	S	N	27.55	2	156	92	22	0.8	N	9.83	2.9	4.5	9.43
59	Mr.Rajiv	994	42	M	-	Y	Y	NS	N	28.52	2	144	92	17	0.8	N	9.46	3.6	4	9.46
60	Mr.Palanivel	1004	67	M	G	Y	Y	NS	N	27.33	2	158	96	30	0.6	N	8.96	3.4	3.7	9.18
61	Mrs.Murugamma	1056	45	F	-	N	N	S	N	25.95	2	148	96	18	0.8	N	9.8	3.9	4.5	9.4
62	Mrs.Renugadevi	1599	36	F	G	N	N	S	Y	24.12	1	152	106	26	0.7	N	10.42	4.0	4.4	10.1
63	Mr.Yuvaraj	1613	39	M	-	Y	N	NS	Y	28.12	2	186	108	29	0.9	N	8.62	3.9	4.4	8.3
64	Mr.Dharmaiah	1646	73	M	G	Y	Y	S	N	21.45	1	166	92	32	1	N	9.62	3.4	3.5	10.02
65	Mrs.Kala	1653	52	F	-	N	N	NS	N	20.42	1	184	108	18	0.8	N	8.64	4.3	4.5	8.24
66	Mrs.Arokiyamary	1655	76	F	-	N	N	S	N	30	2	194	102	28	1	N	8.78	3.3	4.4	8.46
67	Mrs.Vembu	1667	68	F	G	N	N	S	Y	27.2	2	166	116	21	0.7	N	8.36	3.8	3.5	8.76
68	Mr.Selvam	1671	54	M	G	Y	Y	NS	Y	28.04	2	166	98	26	0.9	N	9.03	3.6	3.9	9.11
69	Mr.Sampath	1679	52	M	-	Y	Y	NS	N	20.76	1	194	112	30	0.7	N	8.12	3.4	4	8.12
70	Mr.Johnson	1686	64	M	-	Y	N	S	Y	26.88	2	166	96	24	0.7	N	9.32	3.9	4.2	9.16
71	Mr.Chandran	1687	47	M	-	Y	N	NS	Y	29.75	2	174	104	24	0.8	N	8.91	2.9	4.2	8.75
72	Mrs.Maheswari	1691	42	F	-	N	N	S	N	21.37	1	162	96	21	0.8	N	9.37	4.1	4	9.37
73	Mrs.Fathima	1958	55	F	-	N	N	S	Y	20.99	1	164	98	20	0.7	N	8.66	4.2	4	8.66
74	Mr.Rajendran	1968	45	M	G , P	N	Y	NS	Y	26.91	2	142	92	19	0.7	N	10.5	3.7	4.6	10.02
75	Mr.Abraham	1970	46	M	H , S	N	N	S	Y	28.69	2	158	96	22	0.8	F	10.48	3.9	4.36	10.19

MASTER CHART

ESSENTIAL HYPERTENSION PATIENTS (STUDY GROUP/ GROUP - A)

S.No	NAME	Op/Ip No	Age	Sex	Symptoms	Smoking	Alcohol	Life style	Family H/O	BMI (Kg/m^2)	BMI Group	BLOOD PRESSURE(mmHg)		Blood Urea (mg/dl)	Serum Creatinine (mg/dl)	USG Abd	T. Sr. Calcium (mg/dl)	Serum Phos (mg/dl)	Serum Albumin (g/dl)	C Sr. Calcium (mg/dl)
												Systolic	Diastolic							
76	Mrs.Rambika	1972	55	F	H	N	N	S	N	27.7	2	150	94	20	0.7	N	10.23	3.5	4.33	9.96
77	Mr.Manoharan	1975	36	M	-	Y	Y	S	N	27.71	2	150	108	29	0.9	N	9.6	3.7	4.8	8.96
78	Mrs.Panchali	14849	65	F	G	N	N	NS	N	25.51	2	188	108	26	0.8	N	9.8	3.9	4.4	9.48
79	Mrs.Baby	21468	42	F	G , H	N	N	S	Y	28.12	2	146	104	18	0.7	N	9.52	4.1	4.6	9.04
80	Mrs.Ramani	22523	55	F	-	N	N	S	N	34.31	2	174	108	27	0.9	N	8.9	3.9	4	8.9
81	Mrs.Kotteswari	24141	55	F	G , S	N	N	S	N	31.11	2	170	104	26	1.1	N	8.72	3.8	4.4	8.4
82	Mrs.Padmavathy	24222	50	F	H , G	N	N	NS	N	23.98	1	204	122	26	1	N	7.8	4.1	4.3	7.56
83	Mr.S.Durai	24270	54	M	EF	Y	Y	S	N	19.66	1	194	116	20	0.1	N	10.13	3.9	4.33	9.87
84	Mrs.Amudha	24411	44	F	G , F	N	N	NS	Y	34.58	2	196	120	22	0.9	F	8.5	3.9	4.6	8.02
85	Mr.Kannaiah	24423	85	M	-	N	N	S	N	19.42	1	152	92	32	0.9	N	8.95	3.4	4.2	8.79
86	Mr.M.Durai	24441	60	M	H , G	Y	Y	NS	Y	27.48	2	174	108	20	0.7	N	8.3	4	4.8	7.66
87	Mr.Mahadevan	24465	65	M	G	Y	N	S	N	24.11	1	150	96	30	1	N	8.79	3.3	4.3	8.55
88	Mr.Shankar	24502	61	M	G	N	N	NS	N	27.79	2	158	98	24	1	N	9.66	4	4.14	9.55
89	Mr.Raju	24532	55	M	G	N	Y	S	N	23.62	1	190	122	24	1.1	N	9.01	3.9	3.54	9.38
90	Mr.Karunakaran	24533	50	M	-	Y	Y	S	N	21.75	1	186	106	24	0.9	N	9.55	4.2	4.11	9.46
91	Mr.Raja Bagadhur	24615	75	M	G , S	N	Y	S	N	22.14	1	152	92	23	1.1	N	9.04	4.1	3.51	9.43
92	Mr.Munusamy	24696	55	M	-	N	Y	NS	N	26.42	2	152	92	34	1.1	N	9.46	3.9	4.24	9.27
93	Mrs.Angamma	24737	41	F	H	N	N	NS	N	22.22	1	178	108	24	0.8	N	9.25	4.0	4.04	9.25
94	Mrs.Jeyalakshmi	24802	63	F	G	N	N	S	Y	26.99	2	208	96	18	0.8	N	9.16	3.6	4.4	8.84
95	Mr.Srinivasan	24820	56	M	G	N	N	S	N	20.43	1	168	100	40	1.1	N	9.51	3.3	3.87	9.61
96	Mr.Kannan	25126	65	M	-	Y	Y	NS	N	29	2	164	102	22	0.8	N	9.48	4.5	4.3	9.24
97	Mrs.Jayanabee	25143	80	F	-	N	N	S	N	20.41	1	152	102	24	1.1	N	9.68	4.6	4	9.68
98	Mr.Veerasamy	25365	45	M	G	N	N	NS	Y	23.66	1	184	102	18	0.7	N	8.3	3.4	4.9	7.58
99	Mr.Santhanam	40201	49	M	G , E	Y	Y	NS	Y	21.64	1	170	112	24	0.9	N	9.61	3.1	4.22	9.43
100	Mr.Narayanan	40884	41	M		N	Y	NS	Y	20.76	1	196	96	29	0.9	N	8.75	4.7	3.9	8.83

MASTER CHART

NORMOTENSIVE INDIVIDUALS (CONTROL GROUP/ GROUP - B)

S.No	NAME	Op/Ip No	Age	Sex	Smoking	Alcohol	Life style	Family H/O	BMI (Kg/m^2)	BMI GROUP	BLOOD PRESSURE(mmHg)		Blood Urea (mg/dl)	Serum Creatinine (mg/dl)	USG Abd	T . Sr. Calcium (mg/dl)	Serum Phos (mg/dl)	Serum Albumin (g/dl)	C. Sr. Calcium (mg/dl)
											Systolic	Diastolic							
1	Mrs.Parimala	2045	72	F	N	N	S	N	28.62	2	110	70	27	0.8	N	8.34	3.8	4.5	7.94
2	Mr.Shanmugam	2789	46	M	Y	N	NS	Y	20.21	1	122	76	22	1.1	N	10.6	3.5	3.8	10.76
3	Mrs.Krishnaveni	2980	47	F	N	N	NS	N	30.45	2	116	80	24	0.9	N	8.7	3.9	4.5	8.3
4	Mr.Sivakumar	3065	62	M	Y	N	NS	N	20.64	1	112	72	29	0.7	N	11.74	3.5	4.3	11.5
5	Mrs.Pachaimmal	3228	56	F	N	N	NS	N	27.82	2	110	72	36	1	N	9.72	3.6	3.7	9.96
6	Mr.Kuppusamy	4667	68	M	Y	Y	S	N	20.3	1	100	64	22	0.9	N	10.08	3.8	4.3	9.84
7	Mr.S.Madavan	4779	42	M	N	Y	NS	Y	29.75	2	106	60	30	0.1	N	8.08	2.9	3.6	8.4
8	Mrs.Devi	5039	42	F	N	N	NS	N	19.35	1	120	76	28	0.8	F	10.92	3.2	4	10.92
9	Mrs.Thangamma	5057	66	F	N	N	NS	N	28.13	2	110	72	36	0.7	N	9.88	3.2	4.2	9.72
10	Mr.Murugappan	6234	58	M	Y	Y	NS	N	28.12	2	120	76	28	0.8	F	9.12	3.5	4.5	8.72
11	Mrs.Andalamma	6785	58	F	N	N	S	Y	21.37	1	106	60	30	0.1	N	9.68	3.8	4.1	9.52
12	Mrs.Rashida	7889	58	F	N	N	S	Y	28.12	2	100	64	22	0.9	N	9.46	4.1	4.1	9.38
13	Mrs.Kanagavalli	10112	57	F	N	N	NS	N	27.96	2	106	60	30	0.1	N	9.54	3.8	4.4	9.22
14	Mr.Krishnamoorthy	12354	44	M	N	Y	NS	N	22.22	1	120	76	28	0.8	N	10.9	3.9	3.9	10.82
15	Mr.Ramalingam	12387	62	M	Y	Y	S	Y	23.18	1	110	70	27	0.8	N	9.46	3.4	5.2	8.5
16	Mrs.Shanthi	13657	46	F	N	N	S	Y	27.55	2	112	72	29	0.7	N	10.96	3.0	4.4	10.64
17	Mr.Mohammed	13678	56	M	Y	Y	NS	N	20.76	1	112	70	23	0.6	N	9.7	3.9	3.9	9.78
18	Mrs.Mary	13998	64	F	N	N	S	N	26.88	2	106	74	28	0.7	N	9.94	3.9	4.3	9.67
19	Mr.Raman	14367	59	M	Y	Y	S	Y	20.95	1	122	76	22	1.1	N	9.94	4.0	3.9	10.02
20	Mr.Murali	14546	48	M	Y	N	S	Y	20.07	1	120	78	26	0.8	N	9.7	3.9	4.1	9.62
21	Mr.Mariappan	14876	46	M	Y	N	NS	Y	27.69	2	116	80	24	0.9	N	10.36	3.8	4.2	10.2
22	Mrs.Indirani	15243	42	F	N	N	S	N	26.77	2	100	64	22	0.9	N	9	4.1	3.5	9.4
23	Mr.Anwar basha	15678	74	M	N	Y	S	Y	28.33	2	110	72	36	1	N	9.92	4.3	4	9.92
24	Mrs.Amudha	18456	48	F	Y	N	S	Y	19.38	1	100	64	22	0.9	N	9.12	4.0	4.2	8.96
25	Mr.Raja	18458	49	M	N	Y	NS	N	28.67	2	120	76	28	0.8	N	11.13	4.0	3.7	11.4

MASTER CHART

NORMOTENSIVE INDIVIDUALS (CONTROL GROUP/ GROUP - B)

S.No	NAME	Op/Ip No	Age	Sex	Smoking	Alcohol	Life style	Family H/O	BMI (Kg/m^2)	BMI GROUP	BLOOD PRESSURE(mmHg)		Blood Urea (mg/dl)	Serum Creatinine (mg/dl)	USG Abd	T . Sr. Calcium (mg/dl)	Serum Phos (mg/dl)	Serum Albumin (g/dl)	C. Sr. Calcium (mg/dl)
											Systolic	Diastolic							
26	Mrs.Ponniammal	18976	68	F	N	N	NS	N	30.14	2	106	60	30	0.7	N	9.72	3.8	4	9.72
27	Mrs.Shankari	19234	51	F	N	N	S	N	21.8	1	110	70	27	0.8	N	9.24	4.3	3.9	9.32
28	Mr.Chinnappan	19657	58	M	Y	N	NS	N	27.33	2	112	72	29	0.7	N	9.2	4.1	4.2	9.04
29	Mrs.Sangeetha	19890	49	F	N	N	NS	Y	19.6	1	112	72	29	0.7	N	9.3	3.8	4	9.3
30	Mr.Arokiyasamy	20023	56	M	N	Y	S	N	21.99	1	116	80	24	0.9	N	10.82	3.5	4.2	10.66
31	Mr.James	20442	46	M	Y	Y	S	N	22.49	1	120	76	28	0.8	F	10.26	4.1	4.2	10.1
32	Mr.Suppaiah	20897	75	M	Y	N	NS	N	19.38	1	110	70	27	0.8	N	10.1	3.9	3.5	10.5
33	Mr.Srinivasan	21309	64	M	N	N	NS	N	27.33	2	106	60	30	0.6	N	8.2	3.7	4.5	7.8
34	Mr.Karthik	21829	47	M	Y	Y	S	N	28.12	2	110	72	36	1	N	9.46	3.5	4.1	9.38
35	Mr.Ismail	21952	47	M	N	Y	NS	N	29.06	2	122	76	22	1.1	N	9.96	3.9	4.1	9.88
36	Mrs.Meena	22609	46	F	N	N	NS	N	28.12	2	112	72	29	0.7	N	9.64	3.6	3.8	9.8
37	Mrs.Vasantha	24421	65	F	N	N	S	N	22.65	1	110	72	36	1	N	8.79	3.8	3.2	9.43
38	Mrs.Bakkiyam	24838	45	F	N	N	S	N	28.62	2	106	74	28	0.7	N	8.45	4.4	3.4	8.93
39	Mrs.Kamala	25148	56	F	N	N	NS	N	26.04	2	116	80	24	0.9	N	10.28	4.3	4	10.28
40	Mrs.Chandra	25149	52	F	N	N	NS	Y	23.76	1	122	76	22	1.1	N	9.68	3.9	3.8	9.84
41	Mrs.Naseer Begum	25183	60	F	N	N	S	N	31.25	2	120	78	26	0.8	N	8.47	4.6	3.9	8.55
42	Mr.Samuel	25675	52	M	Y	Y	S	N	26.88	2	116	80	24	0.9	N	10.16	3.6	4.2	10
43	Mr.Mani	25679	55	M	N	Y	S	N	21.12	1	122	76	22	1.1	N	9.2	3.8	4.5	8.8
44	Mrs.Padmini	25987	78	F	N	N	S	N	20.8	1	110	72	36	0.8	N	9.1	2.9	3.5	9.5
45	Mr.Lakshman	27456	64	M	N	Y	NS	Y	27.94	2	106	60	30	0.1	N	9.68	3.2	4.2	9.52
46	Mrs.Rukhmani	27865	56	F	N	N	NS	N	20.95	1	100	64	22	0.9	N	10.96	3.2	4.8	10.32
47	Mrs.Manjula	28134	36	F	N	N	NS	N	28.52	2	110	72	36	0.9	N	9.7	3.8	4.1	9.62
48	Mr.Subramani	28675	78	M	Y	N	NS	N	21.45	1	120	76	28	0.8	F	10.01	4.1	4.3	9.77
49	Mr.Sundarraj	40689	52	M	N	Y	S	Y	25.09	2	100	64	22	0.9	N	9.16	4.0	4.2	9.9
50	Mr.Sivakolunthu	40862	27	M	Y	Y	S	Y	21.27	1	106	60	30	0.1	N	9.68	4.3	4	9.68

ABBREVIATION

A I	- Angiotensin I
A II	- Angiotensin II
ACEIs	-Angiotensin Converting Enzyme Inhibitors
AMP	-Adenosine Mono Phosphate
ARBs	- Angiotensin Receptor Blockers
AT_1	- Angiotensin receptor 1.
AT_2	- Angiotensin receptor 2.
ATPase	- Adenosine Tri Phosphatase
BCG	- BromoCresol Green
BMI	- Body Mass Index
BP	- Blood Pressure
Ca^{2+}	- Calcium
CO	- Cardiac Output
CRP	- C-Reactive Protein
DAM	- Diacetyl Monoxime
DASH	- Dietary Approach to Stop Hypertension
DBP	- Diastolic Blood Pressure
ECG	- Electrocardiography
ENaCs	- Epithelial Sodium Channels
H/o	- History of
HT	- Hypertension
ISH	- Isolated Systolic Hypertension
JNC	-Joint National Committee

K^+	- Pottasium
Mg^{2+}	- Magnesium
MI	- Myocardial Infarction
Na^+	- Sodium
NaCl	- Sodium Chloride
NADPH	-Nicotinamide Adenine Dinucleotide Phosphate(Reduced)
NHANES	- National Health And Nutrition Examination Surveys
NO	- Nitric Oxide
NOS	- Nitric Oxide Synthase
PCR	- Protein Creatinine Ratio
PIH	- Pregnancy Induced Hypertension
PR	- Peripheral Resistance
PTH	- ParaThyroid Hormone
RAAS	- Renin-Angiotensin-Aldosterone System
RBC	- Red Blood Cells
SBP	- Systolic Blood Pressure
TGF-β	- Transforming Growth Factor – Beta
WHO	- World Health Organization
cm	- Centimetre
dl	- Decilitre
g	- Gram
hr	- Hour
Kg	- Kilogram

m^2	- Metre square
mg	- Milligram
min	- Minute
ml	- Millilitre
mm^3	- Cubic millimetre
mmHg	- Millimeters of Mercury
nm	- Nanometre
Yr	- Year
µL	- Microlitre
α	- Alpha
β	- Beta
%	- Percentage
&	- And
<	- Less than
>	- More than
±	- Plus or minus
≤	- Less than or Equal
≥	- More than or Equal

MASTER CHART – ABBREVIATION

S.No	- Serial Number
Ip/Op No:	- Inpatient/Outpatient Number
Age:	
Sex:	M – Male ; F – Female
Symptoms:	G – Giddiness
	H – Headache
	EF – Easy Fatigability
	S – Syncope
	E – Epistaxis
	P – Palpitation
Smoking:	Y – Yes ; N – No
Alcohol:	Y – Yes ; N – No
Lifestyle:	S – Sedentary ; NS – Nonsedentary
Family History:	Y – Yes ; N – No
BMI:	Body Mass Index
BMI Group:	1 – BMI < 25 ; 2 – BMI > 25
mmHg:	Millimeters of Mercury.
USG – Abd:	N – Normal ; F – Fatty liver
T.Sr.Calcium:	Total Serum Calcium
Serum Phos:	Serum Phosphorus
C . Sr. Calcium:	Corrected Serum Calcium

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